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Section of Epidemiology and State Medicine.

President—M. GREENWOOD, F.R.C.P., F.R.S.

[*October* 23, 1931.]

PRESIDENT'S ADDRESS.

By MAJOR GREENWOOD, D.Sc., F.R.C.P., F.R.S.

A PRINCIPAL—perhaps indeed the only—justification of a presidential address is the opportunity it affords of exploring a subject which would not be suitable for a regular paper but is of interest to members of the Section. The recent and regretted retirement from official work of Dr. T. H. C. Stevenson, our Jenner Medallist, makes it not inappropriate that we should give some consideration to the history of the institution he served so well and even, perhaps, speculate a little on its future.

The General Register Office is a wonderful institution; how wonderful it is, possibly even some of our Members do not realize. Perhaps it would not be too Macaulayesque to assert that the publications of that curious department between 1850 and 1880 contained more valuable contributions to our knowledge of the theory and practice of vital and medical statistics than were made by all other contemporary official and private investigators throughout the world. Even now, when there are so many efficient Government statistical organizations and so many unofficial statisticians and when, as we are assured by so many intelligent foreigners and natives, the English are stupid and uneducated, Somerset House contrives to tell us a great deal more about the way these stupid people live and die than the staff of the *Statistique Générale de la France* seem to be able to discover about their clever countrymen.

I suppose my fellow members of the Fabian Society might use the General Register Office to point a moral and adorn a tale. Not one of the pioneers of statistical theory in the 18th century was an Englishman; the greatest of them were Frenchmen. What names have we to put beside De Moivre and Laplace? True, at the end of the 17th century we had John Graunt, but it was left to foreigners to realize the importance of Graunt: and a century ago a comparison between England and the Continent in statistical matters would not have favoured us. An Act of Parliament was passed, a new branch of the civil service created and our trained bureaucracy produced something which has become the envy of the world. How different is all this from the corrupt individualism of the 18th century! How unlike Edmund Burke's account of the Board of Trade, that Board which, you will remember, "had both its original formation and its regeneration in a job. In a job it was conceived and in a job its mother brought it forth."

The story of the General Register Office, if I have read it aright, is not, however, quite so simple; it is much more human. It does indeed illustrate some of the fine virtues of bureaucracy, but it also illustrates how extraordinarily advantageous to the public jobbery may sometimes be, for if the Office was not actually conceived in a job, its enormous success is certainly due to two jobs, a little job and a bigger

job. I take it we shall agree to define a job as the appointment of a person to an office, not because there is good evidence that he is a suitable person to hold that office, but because he is favoured by somebody who can influence the appointment. That was how our two greatest military commanders obtained their chances, John Churchill and Arthur Wellesley.

That, I fancy, was how the creators of English Vital Statistics got *their* chances. Major Graham was appointed Registrar-General by his brother, the Home Secretary; William Farr was appointed compiler of abstracts through the good word of the Court Physician, Sir James Clarke. Better appointments could not have been made, but nobody knew that when they were made or, so far as appears, tried to find out. Both appointments were jobs, and, in the words of the Savoyard, good jobs too. One of these days somebody will write a life of William Farr, and it should be a very interesting book. The author will probably be a candidate for the degree of Ph.D. of the University of Columbia and will have read everything which has ever been written by or about Farr. He will even have read this presidential address. In anticipatory gratitude I will give him the opportunity of correcting the errors of a former president of the Section of Epidemiology and State Medicine of this English Society by printing an impression of Farr based on no deep research.

William Farr was born in the hamlet of Kenley in Shropshire on November 30, 1807. His father, John, was a farm labourer who, with his wife, Katherine, migrated to the village of Dorrington, a few miles from Shrewsbury, soon after William's birth. Here they entered the service of a retired cab proprietor of Bath named Pryce, apparently much on the terms one would expect a married couple to live in the service of an elderly bachelor of moderate private means. Farr had two brothers and a sister, all of whom predeceased him—one died a cooper in his native village, two entered the coach-building trade and the sister died a dressmaker in Shrewsbury. Farr's mother died in 1845, aged 65, and his father in 1864, aged 81. William was the pet of the old cab proprietor, who paid for his education. This was desultory. An early biographer, Mr. F. A. C. Hare, who published a pamphlet in the year of Farr's death, asserted that Farr was sent to Shrewsbury School, but produced no evidence (the point is of some importance as Shrewsbury School, then under Samuel Butler, was a famous educational establishment), and I have little doubt that Noel Humphreys was right in his statement that William never had any regular schooling at all, was never sent to any but a local school—the master of which was "idle and empty-headed"—and only set in the way of earning a living in his nineteenth year. Not having received a "regular" education, William's only choice of a professional career lay between being an attorney or an apothecary and he choose the latter. That usually meant going to live in the family of a general practitioner, where he might have learned something about general practice and acquired some worldly wisdom—knowledge of the second kind he never subsequently acquired; his loss, to the tune of some thousands of pounds, and our gain. Actually it meant formal indentures to a general practitioner and real pupilage to an erudite physician in Shrewsbury. During the three years, from 1826 to 1829, that William Farr trotted backwards and forwards seven miles to Shrewsbury to Dr. Webster he laid the foundations of that curious, unsystematic knowledge which was a good deal more use to him than even Westminster and Trinity College, Cambridge, would have been.

The self-educated man usually misjudges his relation to others who have enjoyed the advantages (and suffered the disadvantages) of an academic education. He will either over-estimate or under-estimate what others know. If schoolmasters and professors are of any use at all—and, as a strict trade unionist, I can only answer that question in one way—their pupils acquire some kinds of knowledge more easily than the self-taught. The latter, not remembering this but remembering that *they*

acquired knowledge with painful effort, may easily suppose that others would not take the trouble they themselves took, and remain ignorant. But, on the other hand, the self-taught might, supposing, for argument's sake only, that such a thing were possible, over-rate the value of professors and imagine that their pupils knew much the self-taught were ignorant of.

Farr tended a little to the former error. Perhaps his contemporary, Macaulay, might have said of him as of the author of Haji Baba, that he was fond of quoting passages of Latin which everybody else knew and didn't trouble to quote. But Macaulays are rare, and quite nicely educated people have read and remembered much less than Farr. Another disadvantage, or, depending upon the point of view, advantage of self-education is that it encourages general reading and distracts one from the main chance. When Farr's benefactor died and left Farr, at the age of 21, a legacy of £500, Farr did not behave at all prudently. Instead of immediately setting about to acquire some really impressive letters to add to his name he behaved like a Renaissance scholar, wandering about the Continent learning things, instead of collecting diplomas. Not until four years after receiving his legacy did he sit for any examination at all, viz., that for the L.S.A., and then, after another year and taking a wife, he set up in practice at 8, Grafton Street, Fitzroy Square. I hope I may live to read the American documented biography of the six years between Farr's setting up as a doctor in Grafton Street and his appointment to the General Register Office in 1839. Even 100 years ago it cost money to house, feed and clothe a young doctor and his wife. That a country lad with no known connections in town, and very ordinary professional qualifications, would do much in practice seems unlikely, particularly if he advertised lectures in his private house on so mysterious a subject as "Hygeine" (so spelt). Two such lectures were printed in *The Lancet* of 1835-6. One deals mainly with the sanitary code of Moses, the other with Hippocrates. Old Mr. Pryce's legacy must have been spent by then, and another legacy of £500, from the Shrewsbury physician, did not fall in until 1837. There is printed evidence (in the shape of a letter to *The Lancet*) that Farr had *one* patient. There is evidence of professional journalism—e.g., a favourable notice in *The Lancet* of a Medical Almanack edited by him, and of course his contribution to McCulloch's book and his work for Sir James Clarke—but none of remunerative work. His young wife died of consumption in 1837 and it is not *very* speculative to suggest that those six years were very hard years indeed. According to Hare, Farr let lodgings and had at least one resident pupil. But Farr did attract patrons, first old Mr. Pryce, next Dr. Webster of Shrewsbury, then Thomas Wakley, and finally Sir James Clarke. Wakley, the Cobbett of the medical profession, counts, I fancy, for a great deal in the story. I cannot find any indication that he employed Farr as a paid journalist, but he not only printed his lecture on "*Hygeine*," but a long paper on Life Assurance and a curious essay on the clinical statistics of cholera, while at the end of a favourable notice of Farr's essay in McCulloch, the following sentences are printed in leading type: "The pages of this Journal have frequently been enriched by statistical contributions of Mr. Farr, and we feel much pleasure in recording our approbation of the article now before us which cannot fail to lay a lasting foundation of honour for its learned author." (*Lancet*, 1838-39, ii, p. 551).

Wakley was a formidable person; his cut-and-thrust style was different from Farr's; one cannot think that Farr's strong points would have appealed much to him, and Farr's weak points would have irritated him. But he was a generous man, fighting the battle of the under-dog against the Mandarins (in our slang) or Bats (in his); possibly the circumstances of the patientless L.S.A. touched his heart. There is much disinterested kindness in the world. Perhaps Wakley did Farr another good turn by introducing him to the notice of Dr. James Clarke, who was not likely to be in the way of meeting L.S.A.'s in Grafton Street. At least Dr. James Clarke did employ Farr to help him statistically in the preparation of a book on consumption

which was published in 1835. That very year Clarke became physician to the Duchess of Kent and in two years Sir James Clarke was physician to Her Majesty and, for a time, a very important person indeed. It was probably Sir James's good word which secured Farr's appointment on 10th July, 1839, as compiler of abstracts in the new General Register Office, which he was not to leave until February 1st, 1880.

Although £350 a year, Farr's initial salary, would have been equivalent to at least £600 now, neither its amount nor the title of his post suggests that Mr. Farr was officially any more important than the three young gentlemen in the Internal Navigation Office of *The Three Clerks*, and (until the definitive biography appears) I am ignorant of his relation to the head of the office. However, from the first, Farr's hand can be seen. Indeed, in the second of the annual reports appeared his first note on the "law" of epidemics, the significance of which was pointed out by our lamented colleague John Brownlee.

But the reports of the First Registrar-General, T. H. Lister, are in the restrained manner which we are accustomed to associate with blue books. The fourth annual report, the first signed by Major George Graham, the Second Registrar General, and to be Farr's official chief to the end, is also perfectly correct, although a reference in it to the valuable assistance of Mr. Farr has a temperature rather above that of 4° centigrade—the temperature, my erudite hearers will remember, at which water attains its maximum density, and therefore the proper temperature of strictly official documents. But between the issue of the fourth and the fifth annual reports something happened, some psychological revolution was effected in the General Register Office.

I picture to myself an earnest statesman of the year 1843, Gladstone, perhaps, in his serious youth on the verge of the Cabinet, opening that Annual Report and finding on p. xvii a properly official table of figures; turning to the opposite text for a sober explanation, his eye falls upon these words:—

"The mental faculties, ripened and developed by experience, will not protect the frame from the accelerated and insiduous progress of decay; the toil of the labourer, the wear and tear of the artisan, the exhausting passions, the struggles and strains of intellect and, more than all these, the natural falling off of vitality, will reduce the numbers to 9,398 by the age of eighty. Here we may pause a minute."

We may indeed; the affrighted statesman's eye will travel down the page and be arrested by a Latin quotation. He reads:—

"After the age of 80 the observations grow uncertain; but, if we admit their accuracy, 1,140 will attain the age of 90; 16 will be centenarians; and, of the 100,000, one man and one woman—like the lingering barks of an innumerable convoy—will reach their distant haven in 105 years, and die in 1945. Crebescunt optatæ auræ, portusque patescit—Jam propior."

"*Can* this be the *Registrar General* speaking, the brother of my esteemed senior the Rt. Hon. Sir James Graham?" he will ejaculate. He will find that the report, which makes a cursory allusion to some appended valuable remarks by Mr. Farr, is subscribed

"I have the honour to be, Sir,
Your faithful Servant,
GEORGE GRAHAM."

Perhaps he may close the volume with the thought that a rather valuable piece of official patronage will no doubt soon be available.

He will be wrong. In 1846 the Home Secretary's faithful servant is found deploring "little children" brought up "in unclean dwellings and impure streets"

and "left alone long days by their mothers, to breathe the subtle, sickly vapours—soothed by opium, a more 'cursed' distillation than 'hebenon'." In the report on 1853, the Registrar-General, in lighter vein, addressing Sir George Grey (a man of vast and curious erudition) adverts to the curious fact that one person in 73 of the population is a Smith, but only one in 174 is a Brown, and appends—"as a matter of curiosity rather than of practical use"—a four-page list of peculiar surnames, such as Cabbage, Earwaker and Puddle (the fictionally classical names of Bultitude, Smallpiece and Twist are here). To the end, the Registrar-General's official interests and literary style are very unlike those of most highly placed civil servants and curiously similar to those of Mr. Farr. In fact the reports of the Registrar-General and, after 1841, of the Census Commissioners, are evidently the compositions of Farr. Possibly Graham made with his subordinate an agreement on the lines of that between Frederick the Great and his people. Graham should do what he thought proper, and Farr should write what he pleased. An admirable arrangement, for there is reason to believe that Graham was a thoroughly competent business man, and evidence that Farr was not a business man at all. We sometimes forget that the *statistics* of the General Register Office are a by-product, and that the public at large is much more interested in obtaining copies of birth, marriage and death certificates for personal uses than in calculations. Unless that business *is* properly organized and administered, the public will not be satisfied with calculations, however ingenious. George Graham will never find a *sacer vates*, not even in America; but George Graham made Farr possible.

The letting loose in this way of an inquisitive, widely-read enthusiast, with a *flair* for the meaning of statistical statements, was a splendid piece of luck. How he organized our vital statistics I need not tell you, that is part of the common knowledge of educated men. How directly and indirectly—through Simon—he stirred the public conscience you also know. Perhaps his curious little hunts of statistical hares—save that of the elusive hare which is the epidemic "law"—are less known. A young medical statistician in want of a subject for research should read the Annual Reports steadily; he will find enough suggestions for a wilderness of theses.

When Farr retired it must have been difficult to choose a successor. His immediate successor, William Ogle, had attainments which Farr might have envied. I think Farr really did enjoy the classics, at least the Latin classics, but he was no more a "scholar" than I am, and would have been almost as incapable as I am of fulfilling Macaulay's definition of a scholar, viz., a man who can read Plato with his feet on the mantlepiece. Ogle could have done that easily; he was an exact scholar whose memory will be kept green by Aristotelians rather than statisticians. Not that Ogle was an incompetent statistician; on the contrary. He was the originator of corrected or, as we now say, standardized death-rates. But nobody can deny that in Ogle's time the individuality, quaintness, if you like, which made the publications of the General Register Office so attractive, evaporated. Ogle's successor, Tatham, a competent statistician, with as much more knowledge of sanitary administration than Farr as Ogle had of Greek literature, did not recapture the spirit of the old days. A quarter of a century after Farr's death, the reports of the General Register Office had completely reverted to the standard official literary style. They were correctly written, there were no quotations from Ariosto or Vergil; they were informative, but certainly not lively. Then began the epoch which, to our sorrow, is now closing, and Dr. T. H. C. Stevenson occupied Farr's chair. The spirit of Farr was recaptured. I do not mean that Dr. Stevenson wrote like Farr. I think he would have been very unwilling to do so, and I do not recall a single quotation from any Latin or Italian classic in his "letters." His indictments of public health conditions have been more restrained in language, but not less deadly in effect than Farr's. He has hunted fewer hares,

but the hares he has hunted have been captured. Like Farr he has been interested —if I may use a Farr-like metaphor—in the people going up and down to Camelot, whose shadows appeared in the mirror of Shalott, set up in the Strand. He has never said anything about opiates being more cursed than Hebenon, but he has, year by year, told the story of infant mortality in a way to inform our minds and touch our hearts. A reprint of Dr. Stevenson's "letters," with the slightest of re-arrangements and a few connecting paragraphs would be a model handbook of medical statistics. He can lay down his office with the consciousness that future historians will rank him but little after the founder of modern medical statistics.

The addition of the name of Stevenson to the roll of Jenner Medallists of this Society, a roll which included such names as Manson, Laveran and Shirley Murphy, is a peculiarly appropriate recognition of high scientific achievement.

The achievements of Farr and Stevenson have made the continuance of their particular kind of work difficult, perhaps impossible. I do not mean that a man of Farr's or of Stevenson's ability, having that insight into the real meaning of statistical data, which is an innate quality, not an acquirement which mathematical training can confer, and given a free hand in the General Register Office, might not make equally important discoveries. I do mean that it is now much more difficult to give an official vital statistician his head in the way Graham gave Farr his head. Eighty years ago, politicians and other ephemerally important folk did not really take medical statistics seriously. Not even a Graham would have ventured to permit a Farr to career about the field of economic statistics hitting a head here, halloing on a mob there, as he did in the vital-statistical arena. Statesmen and business men would have been seriously alarmed and have protested that official sanction ought not to be given to these wild ideas. The news value, the tendentious interpretation, of medical and vital statistics can no longer be ignored. It is possible that an official department may be compelled to restrict the limits within which individual members of its staff can have a free hand for fear of the improper use which may be made of conclusions necessarily imperfect. If a choice has to be made between insecure brilliance and safe dullness, there can be no doubt in which direction official prudence *must* point. But I am not discouraged. We take a pride in our national institutions and the General Register Office is one of our great institutions. I am sure that the present head of that office is proud of its traditions and will do all he can to add lustre to them; I am confident that his successors will live in the same spirit. Something of the personal, human touch may be wanting, but the memory of that enthusiast from Shropshire, who was so odd a civil servant and so English a genius, will be honoured and most of the things he valued will continue to be valued.

Clinical Section.

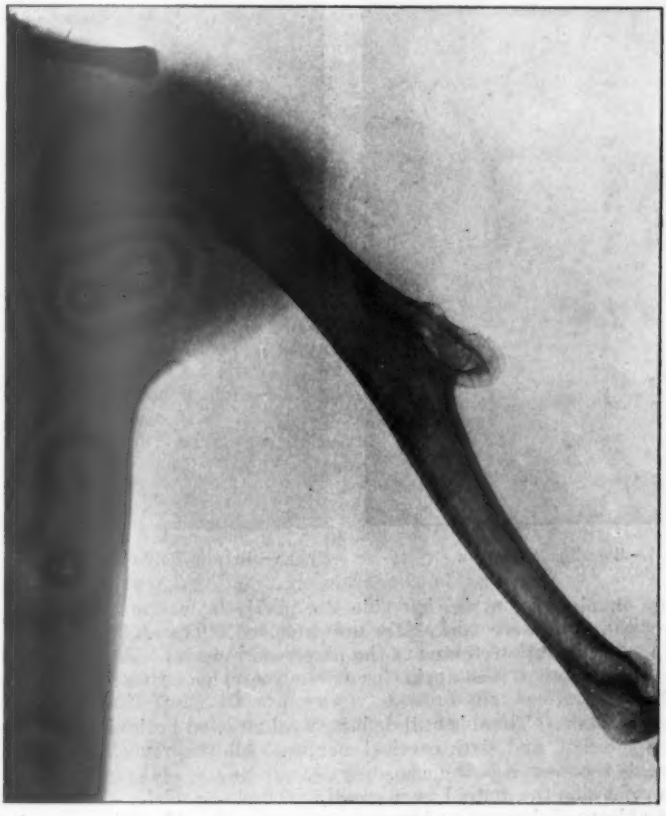
President—Mr. CECIL P. G. WAKELEY, F.R.C.S.

[*October* 9, 1931.]

Multiple Exostoses in a Mother and Two Children.—CECIL P. G. WAKELEY, F.R.C.S. (President).

Mrs. E. E. M., aged 26, has the following exostoses: (1) Inner side of upper end of left tibia. (2) Inner side of right ankle. (3) Fourth left rib, near its costal cartilage. A large exostosis growing from the lower end of the left ulna was removed in Durham in 1918, because it prevented her from using her hand when washing clothes.

There is no history of the condition on either side of the family, but she has two children who both manifest the condition.



Margaret M., aged 5 years.

Skiagram showing exostosis of the humerus.

Margaret M., aged 5, has two large exostoses in the upper third of the left humerus, one on the inner side of the upper end of the right tibia, and one on the outer side of the right fibula at its lower end (see figure).

Clough M., aged 3, has an exostosis on the upper part of the left scapula and another on the outer side of the right fibula. No doubt, as the child grows, other exostoses will be discovered.

Recurring Dislocation of the Right Shoulder-joint. — CECIL P. G. WAKELEY, F.R.C.S.

Norman H., aged 11 years, was first seen at hospital at the age of 3 weeks. He was the second child, and was a month premature. The labour was a difficult one, the presentation being a breech ; the infant had a double Erb-Duchenne paralysis. On the right side all the muscles supplied by the fifth and sixth cervical

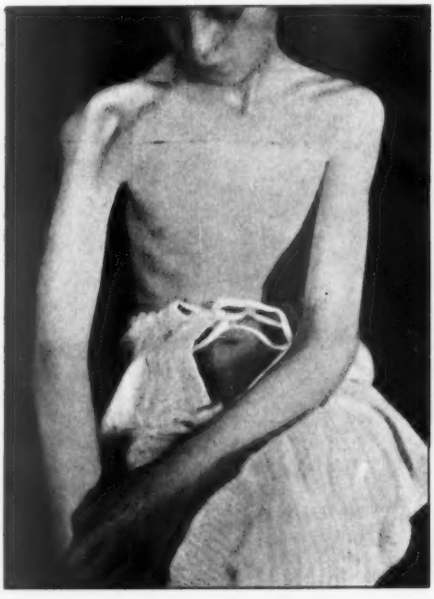


FIG. 1.—Showing dislocation.

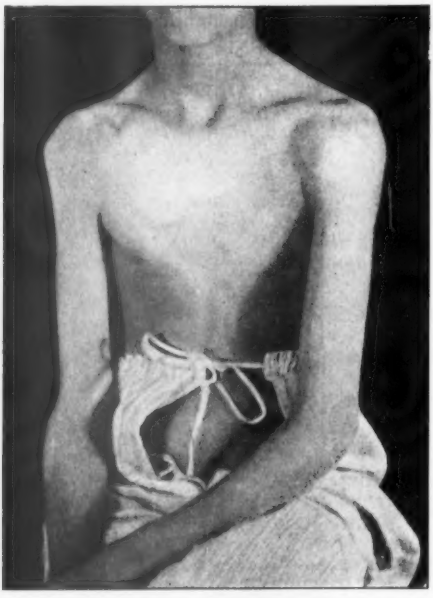


FIG. 2.—Showing head of humerus in glenoid cavity.

nerves were involved ; on the left side the paralysis was not so severe, and the extensors of the wrist were apparently not involved. The child was put up in a double splint to prevent stretching of the paralysed muscles. After the splint had been applied, he was not seen again for eleven years, as he was sent to an orphan home in the country.

Present condition.—There is still definite weakness, on both sides, of the muscles supplied by the fifth and sixth cervical nerves. All the muscles, however, give a normal faradic reaction in both arms.

On the right side the deltoid is markedly wasted, especially in the middle fibres. The boy is able to dislocate the humerus from the glenoid cavity at will, the bone passing backwards towards the spine of the scapula. He can then, by using the pectoral muscles, cause the humerus to retrace its steps into the glenoid cavity again (see figs. 1 and 2). This does not cause any pain.

Skiagrams do not reveal any osteo-arthritic changes in the joint.

An arthrodesis of the joint might be performed, but this should not be until later. The question arises as to the making of new ligaments, or whether a sling operation should be performed.

Mr. H. A. T. FAIRBANK said that in this case an artificial ligament might be made out of the fascia lata; he did not know what other method could be carried out. He wondered whether an oblique ligament from the acromion to the upper end of the humerus, running as far backwards as could be managed, would answer the purpose.

The other alternative would be to put a sling across the back of the head of the bone; but that might interfere with internal rotation. Mr. Wakeley might put in a crucial pair of ligaments—certainly one, running downwards and mostly backwards. That would not interfere with the joint, particularly if it were put in with the arm in 45% abduction. If one were to put the arm up square after the operation, i.e., at 90° abduction, there would be a stretching of the ligament as the arm came down and it might be too loose.

Rupture of the Axillary Vein.—CECIL P. G. WAKELEY, F.R.C.S.

Ernest L., aged 51, a milk roundsman, May 20, 1931, was pushing his barrow in front of him when a motor car struck the barrow head-on from the front, and both his hands and arms were suddenly forced backwards.

Immediately afterwards he felt sudden pain in front of the right shoulder; this continued for two days and has been present ever since but has not been so severe; it is of a throbbing, aching character, and at times shoots down to the wrist. Ten days after the accident, patient noticed a swelling at the root of his neck on the right side, and another below the middle of the right clavicle. These have gradually become larger.

Patient can grip with the right hand as well as before the accident, but on attempting to lift anything he has great pain in the right shoulder. There is a definite flattening over the point of the right shoulder. There are two swellings to be seen on the front of the chest: (1) A round smooth swelling at the base of the right anterior triangle of the neck. It has a definite rounded border, is pulsatile and not tender. The skin over it is normal. (2) A diffuse swelling below the right clavicle and extending towards the right axilla. This is a softish swelling, not tender and non-pulsatile, and it has ill-defined limits.

The right axilla is fuller than the left, especially behind the free outer border of the pectoralis major muscle. The right cephalic vein is prominent, courses across the front of the chest, and passes superficial to the inner third of the clavicle, where it appears to dip down into the lower aspect of the first-mentioned swelling. The venous flow is from the arm towards the neck in this vein. There are also some prominent venules over the inner aspect of the right chest.

Blood-pressure: left arm, 105/70; right arm, 95/70. Both pupils react normally, right pupil is slightly smaller than left.

Skiagrams and screening of chest do not show any abnormality.

I am open to correction as to the title. The swellings are cystic and the lower one is pulsatile. I suggest that the condition is due to extravasation from a ruptured axillary vein, and that there has been a hæmatoma which has become partly absorbed, leaving a cystic condition. If the patient uses the arm the cephalic vein stands out prominently and the arm swells, showing that there is some blockage of its main venous return. Skiagrams do not show anything abnormal in the chest, except a little opacity at the right apex.

Dr. F. PARKES WEBER said he thought the condition was due to a tumour, or else to a cyst connected with some congenital lymphatic abnormality, like "cystic hygroma of the neck" in children. The trauma might have caused hæmorrhage into a tumour or into a lymphatic cyst. It was important to note that it was not before a week after the injury that the main swelling appeared. If it had been an ordinary hæmatoma there would have been diffuse ecchymotic discoloration of the skin.

Renal Dwarfism and Rickets.—BERNARD SCHLESINGER, M.D.

D. B., a boy, aged 7. *Past history.*—Breast-fed. At age of 10 months right foot was noticed to be turned in and patient was treated for rickets with cod-liver oil and artificial sunlight. Treatment continued for some time without improvement. Albuminuria first noticed at age of 4 years. Deformities progressed and child began to complain of thirst and polyuria.

Present condition.—Height, 37 in.; weight, 32 lb. Marked genu valgum and rachitic enlargement of epiphyses of arms and legs.

Blood-pressure, 108 systolic. *Urine*, sp. g. 1015; albuminuria. Microscopic examination shows no casts or abnormal cells. Blood-urea, 36 mgm. per 100 c.c. (on one occasion 80 mgm.); serum calcium, 9·7 mgm.; inorganic phosphorus, 1·7 mgm.; cholesterol, 155 mgm. Urea concentration, 1·5%, 1·8%, 2·3%, one, two and three hours respectively after urea.

Wassermann reaction, negative.

Skiagram.—Marked evidence of active rickets in the upper and lower extremities.

Discussion.—Dr. W. W. PAYNE said he had had two undeniable cases of renal rickets, with a very high blood-urea content. In both the calcium was normal; in one the phosphorus was 2·8 mg. and in the other 3·8 mg. They were not on the high side. He had not had a case of renal rickets with a high phosphorus content. He had had another case analogous to this one, a child, who at 3 years of age, became knock-kneed, the condition having started soon after his second birthday. Skiagrams showed, as in the present case, active rickets. There was a trace of albumin in the urine, but there were no casts. The urine-urea was 2·9, the blood-urea normal; phosphorus low; blood calcium normal. So that in the biochemical findings that case was parallel with the present one. The bony changes occurred later than in ordinary rickets.

Dr. LEONARD FINDLAY said that possibly in this case there was nephritis, in view of the persistent albuminuria and the record on one occasion of a high blood urea, but he would like to know the result of the pigment test which, so far as he was aware, had been positive in all the recorded examples of renal rickets. The dwarfing of the patient strongly suggested renal disease, but the X-ray appearances were just such as were met with in ordinary late rickets. Dr. Schlesinger spoke of the rachitic changes being present from the age of ten months. There might have been rickets at ten months, but there was no way of knowing that the condition had been present ever since. Healing might quite well have occurred and the present mischief be of the nature of a recrudescence. To him (Dr. Findlay), however, the most interesting question in the case, if there was nephritis, was the relationship of the rickets to ordinary late rickets or infantile rickets. Most writers in discussing this question concentrated their attention on the blood changes, and because these were different deduced a different etiology. It should be remembered, however, that the condition of the blood was no criterion of the behaviour of the general metabolism. Had, for instance, the cause of rickets in a child changed because he had developed tetany? Before tetany appeared there was a low blood phosphorus and a normal blood calcium, but when tetany appeared there was a high blood phosphorus and a low blood calcium. In rickets, tetany and cœliac disease with and without rickets, there was a defective and parallel retention of calcium and phosphorus irrespective of the blood-changes. There was no record of a study of mineral balance in renal disease with rickets, but in chronic renal disease there was the same fundamental metabolic change as that met with in rickets. If then there should be shown, as was most probable, the same fundamental change in renal rickets as occurred in all other varieties of the disease, there would seem to be little reason for assuming a different etiology.

A further point of interest in this question was that in chronic renal disease there was a prolonged deprivation of calcium leading to a condition of osteo-porosis. This, we know, renders rickets liable to develop. We have the analogous case of the premature infant, who, being born with a deficient amount of calcium, almost without exception develops rickets. Therefore he suggested that in chronic renal disease there occurred this prolonged calcium starvation, rendering the patient liable to rickets whenever the etiological factor, whatever it was, should come into play. The invalid life of the chronic nephritic rendered him through his confinement indoors likely to be deprived of the action of the sun's rays, and this deprivation was the potent factor in causing rickets.

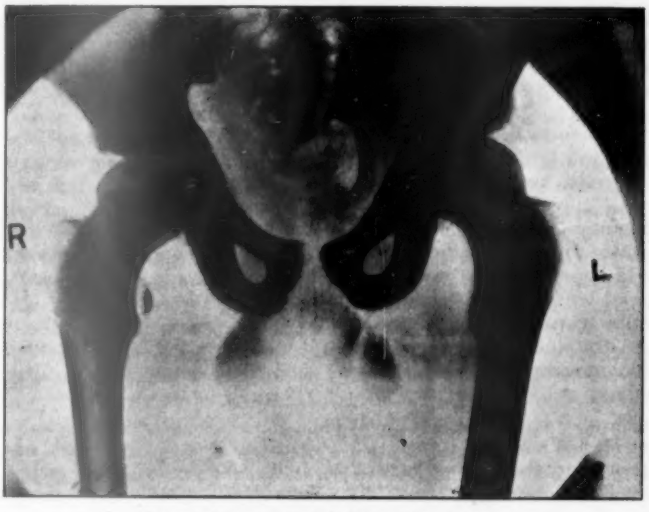
One essential for the development of rickets was growth. Further, if the metabolic change underlying the cause of rickets, viz., the diminished retention of calcium, did not

disappear healing could not result. As chronic nephritis was an incurable and progressive disease one was therefore not surprised that therapeutic measures were of no avail.

Dr. SCHLESINGER (in reply) said that he considered this to be a case of renal rickets. Ordinary rickets could be excluded because the diet during the last four or five years had been anti-rachitic and yet the condition had gradually advanced. He understood that ordinary late rickets only came on after vitamin A and vitamin D deficiency. He did not agree with Dr. Findlay that rickets in renal disease was the same as that produced by a lack of these vitamins. Ordinary rickets was cured by means of ultra-violet light or by supplying the deficient vitamins; renal rickets on the other hand remained unaffected. In fact it had been said that ultra-violet light might prove dangerous and could produce uræmia. It was therefore important to keep a watch on the blood-urea if this treatment were attempted.

Traumatic Separation of the Small Trochanter.—PAUL BERNARD ROTH, F.R.C.S.

Albert B., aged 16, pottery worker, admitted to the Miller General Hospital, 16.7.1931, complaining of pain in the right thigh. The previous day he had been standing on a ladder when his right leg suddenly slipped through and he fell over backwards wrenching his right groin, his knee catching for a moment against the rung above before he slithered backwards down the ladder to the ground. When he got up he found it was very painful to try to walk, and was brought to hospital. When examined lying on a couch he could not lift his right leg; nor could he lift it when allowed to sit up. There was acute deep tenderness in the right groin below Poupart's ligament, at the base of Scarpa's triangle, on the inner side.



Traumatic separation of the small trocanter.

Skiagram (by Dr. Wylie) showed a complete separation of the right small trochanter, with displacement of the fragment upwards one inch. On re-examination, no crepitus could be felt on deep palpation.

The question of operation arose, and it was decided to leave the fragment alone, and treat the condition by rest in bed with the hip and knee semiflexed. After a week, as the tenderness had almost disappeared, patient was allowed to go home.

The result of this treatment has not been good: considerable disability remains. On testing him *lying down* he is now able to lift the limb perfectly, but when *sitting* he has great difficulty in doing so (Ludloff's sign). He states he cannot run, cannot climb a ladder, and has difficulty in swimming. Should the fragment be replaced by operation now?

Mr. H. A. T. FAIRBANK said that he would strongly urge Mr. Roth not to operate in this case. The boy would probably recover without operation. Part of his trouble was due to his having had a nerve-shattering accident. He should have graduated exercises and be encouraged.

Multiple Aneurysms, involving the Thoracic Aorta and the Common Carotid, Subclavian and Axillary Arteries.—B. T. PARSONS-SMITH, M.D.

A. R., female, aged 63.

History.—Aneurysm of the left common carotid artery, 1915; aneurysms of the left subclavian and left axillary arteries, 1916; has had recurring heart attacks—giddiness, breathlessness, rapid beating, etc., during the past two years; now complains of breathlessness and pain in the heart region on exertion, also neuralgic pains (? pressure on the brachial plexus) in the left arm.

Clinical findings.—Heart considerably enlarged downwards and to the left; apex impulse in the anterior axillary line in the seventh space; increased area of dullness at the cardiac base (aortic region), extending two inches to the left and 1½ in. to the right of the midline in the second and third spaces; rhythm regular; systolic and diastolic murmurs at the aortic region; pulse 80, regular; arteries thickened; blood-pressure: right arm 145/60, left arm 140/60; jugular vessels engorged recumbent, and well-marked stasis in the superficial veins over the upper part of the sternum; trachea displaced to the right; skin of the left arm dry and well-marked clubbing of the finger nails of the left hand; Wassermann reaction positive.

Electrocardiogram.—R S complexes of low voltage; flattening of the T waves in all the Leads and typical appearances of preponderance of the left ventricle.

Cases of multiple aneurysms are moderately uncommon, and often only diagnosed at autopsy.

The condition was recognized and well described by physicians as long ago as the eighteenth century, and a certain number of cases have since been recorded, in which the lesions involved the aorta with the mesenteric, the cerebral and various larger arterial trunks.

I have shown this case as exemplifying the relatively slow progress of the disease (the first aneurysm having been diagnosed in 1915) and the long period of comparative freedom from incapacitating symptoms which such patients may very possibly enjoy.

The PRESIDENT said that, clinically, the important feature in this case was the length of time the patient had noticed the swellings. Aneurysms were not now very frequently seen, and those seen before the late war had not such a long history as in this case.

Tumour of Testicle.—ERIC A. CROOK, M.Ch.

H. McQ., aged 31, was admitted to hospital six weeks ago on account of a swelling in the right side of the scrotum that had appeared suddenly without any preceding injury or strain. The condition somewhat resembled a strangulated hernia—a diagnosis that had previously been made—but no intestinal symptoms were present. The opposite testicle was found to be small and imperfectly descended, but the previous condition of the right testicle was not known, neither was the appearance that of a torsion of the cord.

Under treatment for acute orchitis with œdema of the cord, the temperature settled down and the pain and tenderness disappeared. On patient's discharge from hospital, fourteen days later, the swelling was still present, but was distinctly smaller than on admission.

On examination ten days ago the tumour was found to have increased in size and was then painless, and the swelling of the cord gave fluctuation and translucency. Deep pressure in the right iliac fossa revealed a hard irregular swelling along the course of the spermatic vessels.

The condition is now very suggestive to me of malignant growth : it is hard, and is probably spreading along the lymphatics. The Wassermann reaction, ascertained ten days ago, was negative.

Discussion.—The PRESIDENT said that this condition had probably begun as a partial torsion of the cord, and was not a new growth. The swelling passed along the cord as far as the internal abdominal ring.

Mr. CROOK (in reply) said the feature which seemed to be most significant was the hard, knotty irregular cord spreading from the internal ring to the renal region, which he thought was bound to be direct spread of testicular growth. It felt too hard for thrombosis, almost craggy in places.

An Elderly Man, who formerly for many years suffered from Angiitis Obliterans ("Buerger's Disease").—F. PARKES WEBER, M.D.

The patient (M. M.), a Russian Jew, aged 66 (in December, 1931), who, at the age of 25, came to England, was first seen by me in May, 1906. The symptoms had commenced gradually in 1902 or 1903, at the age of 38, with pain in the left lower limb on walking, of the nature of intermittent claudication. The disease progressed by exacerbations, but there were prolonged periods of quiescence, with remission or absence of pain (apart from the intermittent claudication pain). Both lower and the right upper extremities became involved. Besides the typical symptoms, including slight ischæmic ulceration in the feet, he suffered from attacks of superficial thrombophlebitis, notably of the "cutaneous nodular" type.

Since 1924 there have been no active signs of the disease, but the patient has had slight attacks of gout in the big toes and elsewhere, and has developed permanent high blood-pressure with renal involvement. The circulation of blood in the skin of both feet is good, and he can walk slowly for twenty minutes without intermittent claudication. Pulsation is still absent in the dorsal artery of either foot.

The disappearance of all *active* signs of arteritis obliterans in this case seems to me to afford a strong argument in favour of the disease being due to an infective or toxic agent and to be quite distinct from degenerative arteriosclerosis and arteriolosclerosis, though, of course, both the latter may supervene. For further details and references to my earlier accounts of this case, see F. Parkes Weber, "Thromboangiitis Obliterans of twenty-two years' Duration," *Lancet*, 1925, ii, p. 21.

Discussion.—Dr. M. SCHWARTZMAN said that there were certain points of considerable interest in this case. The pulse in the radial artery had been absent, and had reappeared some time ago. It was of course difficult to say whether the recurrence of the radial pulse was due to the re-establishment of the circulation through an artery, which had been in some parts organically obliterated, or to relaxation of an arterial spasm. In some cases of his own, the pulse in the radial artery had been absent, and had then re-appeared after forty-eight hours or so, in response to the administration of muscle-extract which he had suggested, on account of antispastic properties for the treatment of some vascular disease. Such a rapid return of the pulse in the radial or other large arteries was, in some of these cases, most likely to have been due to relaxation of a spasm. If so, that was important from a diagnostic point of view. When one saw a pulseless artery one must not always assume that therefore the artery was organically obliterated, and base on such an assumption the differential diagnosis between the organic and the neurogenic type of arterial disease, or in some cases be guided by it in the choice of the site of amputation.

Secondly, there had been in this present case a disappearance of the red patches when the limb was in the dependent position ("induced erythromelia" according to Buerger's nomenclature), but the patient still showed blanching on elevation of the limb, particularly the right one. This dissociation of postural colour changes was of great significance, and showed that either the minute vessels (the capillary loops and subpapillary venous plexus) had regained their normal tone or the power of oxygen utilization of the tissues had increased.

The suggestion that improvement should not be estimated by "vague clinical signs" but by the oscillometric index was, he thought, entirely wrong. Even with a large artery converted into a fibrous cord there could still be a considerable clinical improvement, which was due to a better blood supply through relaxation of previously spastically contracted small arteries. The patient still suffered from intermittent claudication, and he could not walk more than 200 to 300 yards without stopping. This showed that the intermittent claudication was the last sign to go, not the first. It was to overcome that that the limb had to be supplied with a considerable quantity of blood, whereas a small supply sufficed to overcome other signs, and perhaps even actual gangrene in thrombo-angiitis obliterans. Finally, the condition might become stationary; and this being so, one should condemn early amputation as bad practice and advocate prolonged medical treatment whenever possible.

Dr. PARKES WEBER (in reply) said that the disappearance of the radial pulse could scarcely have been due to spasm, as what drew his attention to it was a local swelling. The swelling was not necessarily due to true thrombo-arteritis; it might have been due to nodular thrombo-phlebitis of venæ comites.

Congenital Jaundice in an Elderly Man—probably belonging to the Congenital Hæmolytic Group.—F. PARKES WEBER, M.D.

A man (G. T. D.), aged 65 (in November, 1931), English. Excepting for considerable, but somewhat variable, jaundice and chronic deafness (due to otosclerosis) and slight nystagmus, he gives the impression of an active, hard-working, healthy elderly man, and has never been seriously ill. I have seen him from time to time during the last fourteen and a half years. Apart from the jaundice and a very strongly positive indirect Hijmans van den Bergh reaction for bilirubin in the blood-serum, he has presented none of the ordinary signs of congenital hæmolytic jaundice (no splenomegaly, no anæmia, no excessive "fragility" of erythrocytes, no excess of urobilin or urobilinogen in the urine), and no family history of jaundice or anæmia can be obtained. Wassermann reaction, negative. For further details and references to my previous accounts of the case see F. Parkes Weber, *Practitioner*, 1930, cxxiv, p. 394.

The case might be summed up as one of "congenital hæmatoidin-jaundice without any other signs of hæmolytic jaundice" (cf. *Med. Press*, 1928, clxxvii, p. 51).

Generalized Sclerodermia.—F. PARKES WEBER, M.D.

The patient (Mrs. C. B.), aged 48, English Jewess, presents a chronic hard diffuse symmetrical thickening of the hands and feet, and, to a somewhat less extent, of the legs (including the lower part of the thighs), the forearms, the face, neck and upper front of the chest. The change involves the skin and subcutaneous tissue and causes more or less limitation of movement in finger and toe-joints, and in ankles, wrists, knees, elbows, and jaws. There is no associated pigmentary or telangiectatic abnormality, excepting hair-like telangiectases over cheeks and nose. The condition developed gradually, commencing seven years ago, together with signs of the approaching menopause. Her fingers tend to be blue in cold weather, and especially when she wakes up in the mornings. Brachial blood-pressure: 170/90 mm. Hg. Dr. Weber regards the case as a typical example of the "puffy" or "hypertrophic" type of generalized sclerodermia or "sclerodactylia," associated, as sclerodactylia often is, with Raynaud-like phenomena. Previous diagnoses have included myxœdema (at first), Raynaud's disease, and subacute dermato-myositis. To support the latter diagnosis Dr. Weber thinks that there ought to have been a history of asymmetrical nodular swellings or symptoms resembling trichinosis

(acute dermato-myositis has been called "pseudo-trichinosis"); moreover, in dermato-myositis there is definite involution of the disease in patients who survive so long as this woman has. At present the patient is having treatment with

Dr. Parkes Weber's case of sclerodermia.

angioxyl and diathermy, as well as massage and radiant heat. Much other treatment has been tried elsewhere.

Addendum.—The last number of *Heart* (1931, xv, pp. 329-350) contains an article by Sir Thomas Lewis and E. M. Landis, in which it is suggested that the sclerodactylia type of generalized sclerodermia is ætiologically connected with the angiospastic symptoms in the fingers. I regard these angiospastic Raynaud-like phenomena as early symptoms of generalized sclerodermia, of the sclerodactylia type. In this connection it may be noted that J. S. B. Stopford (*Lancet*, 1931, ii, p. 782) believes that prolonged narrowing or occlusion of the vasa-vasorum is likely to lead ultimately to arterial thrombosis.—F.P.W.

An Anæmic Breakdown or Crisis in a Child, not connected with definite Congenital Hæmolytic Jaundice—Rapid Recovery.—F. PARKES WEBER, M.D.

The patient, V. F. M., aged 11, an English boy of Italian ancestry, was admitted to hospital late at night on April 25, 1931. The history was that on April 20 he complained of headache and afterwards of pain on the right side of the abdomen. This was followed two days later by much vomiting. Appendicitis had been thought of, but on admission no signs of organic disease were discovered, other than fever (104·2° F.), some dental caries and extreme anæmia (the erythrocyte count was 930,000); no jaundice. There was no history of previous severe pallor or any severe previous illness in the patient, nor of any anæmia or jaundice in the family. Under treatment by blood transfusions (at first) and iron, arsenic and a liver extract, rapid improvement and apparently complete recovery took place. There was no fever after May 13, and the retinal hæmorrhages, which were present at first, gradually disappeared. The spleen could be felt for a time slightly enlarged; no enlargement of lymphatic glands detected. The erythrocyte count, which on May 1 was 780,000, reached 6,400,000 by August 19; a lymphocytosis has been present throughout. There was never any excessive "fragility" of erythrocytes

either in the patient or in his only brother; on September 21, in the patient himself, hæmolysis commenced with the 0·48% sodium chloride solution and was complete with the 0·42% solution. On April 28 the patient's blood-serum gave a strongly positive indirect Hijmans van den Bergh's reaction for bilirubin, and this indirect reaction is still positive (September 21), though not so abnormally strong. Negative Wassermann and Meinicke reactions. Pirquet's reaction (human and bovine): negative. Blood culture (April 28) negative. The urine on April 27 showed a trace of albumin, and gave a positive urobilinogen reaction in the cold, but later on the urine became free from albumin and from excess of urobilinogen.

The case seems to me to be one of temporary anæmic breakdown (crisis of anæmia), of unknown causation in a previously apparently healthy child,[1] perhaps similar in nature to the sudden anæmic breakdowns (accompanied by increased hæmolysis and further enlargement of spleen), which are well known to occur occasionally in the subjects of congenital hæmolytic jaundice. It should be noted that the blood-bilirubin is still slightly excessive (September 21) and that there is still some lymphocytosis.

For the present case I am indebted to my surgical colleague, Mr. H. W. S. Wright (who saw the child previously to admission, when there was a suspicion of appendicitis); and for much help in the examination of the case to my house-physician, Dr. M. Scholtz. Some of the blood-counts are given in the accompanying table.

BLOOD-COUNTS AT VARIOUS DATES.

	April 26	May 1	May 4	June 12	August 19	Sept. 21
Hæmoglobin per cent.	...	19	20	82	92	101
Erythrocytes per c.mm. of blood	930,000	780,000	1,120,000	3,900,000	6,400,000	5,200,000
Colour index	...	1·22	0·9	1·05	0·72	0·97
Leucocytes per c.mm. of blood	10,400	19,800	24,650	5,300	3,500	5,350
Eosinophils per cent.	1	1	0	2	1	2
Basophils per cent.	0	0	0	0	0	0
Myelocytes per cent.	1	3	4	0	0	0
Metamyelocytes per cent. ...	1	3	3	0	0	0
Polymorphonuclear neutrophils per cent.	28	23	19	25	27	37
Lymphocytes per cent.	53	66	71	63	69	56
Monocytes per cent.	16	4	3	10	3	5
Normoblasts seen during differential count of 100 leucocytes	3	7	17	0	0	0
Megaloblasts seen during differential count of 100 leucocytes	4	5	5	0	0	0
Anisocytosis	+++	++	++	+	0	0
Poikilocytosis	+	+	+	0	0	0
Polychromasia	+++	+++	+++	0	0	0
Basophilia punctata	+	...	...	0	0	0
Reticulocytes, per cent. of erythrocytes ...	...	...	13	...	...	0·5
Thrombocytes per c.mm. of blood	...	...	241,920	...	...	982,800

Erythræmia, with Frequent Jaundice, apparently of Hæmolytic Nature. Slow Recovery from Extremely Severe Hæmatemesis.—F. PARKES WEBER, M.D.

The patient (E. F.), aged 42, a married Welsh woman, has suffered for many years from erythræmia, with enlargement of the spleen and liver, and frequently with jaundice of varying degree. There has never been any bilirubin in the urine when it has been examined, and her blood-serum has always given a negative direct and a more or less strongly positive indirect Hijmans van den Bergh reaction ("blood-bilirubin"). I described the case fully in the *British Medical Journal* for November, 1929 (p. 892), and suggested that in this case the jaundice was of hæmolytic nature, possibly a manifestation of ("compensatory") *hypersplenism*; there was no abnormal "fragility" of the red blood-corpuscles.

1 For a possibly similar case see *Klin. Wochenschr.*, Berlin, 1931, x, 1053, report of the demonstration by Kühl of a boy aged 9 years, in full health after a severe attack of anæmia of unknown origin, during which the red cells fell to 800,000 (of which 23,000 were normoblasts) in the c.mm. of blood.

Since then the treatment has been by blood-letting, with some cautious trials of phenylhydrazin. However, the chief treatment has been automatic on the patient's part, by two extremely severe attacks of spontaneous hæmatemesis, the first in January, 1930, the second in January, 1931. From the effects of the latter hæmorrhage the patient has still not completely recovered, as if there were some exhaustion of the hæmopoietic function of her bone-marrow. She is now (September 19, 1931) pale and slightly jaundiced, but states that with this sallow appearance she feels much better and more comfortable than she did when she looked full of blood ("erythræmic"). Her spleen reaches four fingers' breadth, and the liver (in the right nipple line) reaches two fingers' breadth below the costal margin. Her blood-count on August 29, 1931, was: hæmoglobin 45%, erythrocytes 5,400,000, colour-index 0·417, leucocytes 32,800 (eosinophils 5%, metamyelocytes 7%, polymorphs 69%, lymphocytes 18%, monocytes 1%), no nucleated red cells. There was some anisocytosis and slight poikilocytosis. The thrombocyte count, as estimated on May 16, was 1,678,320 per c.mm. of blood. The blood-picture is an excellent example of what I prefer to call *anæmia polycythæmica*, not rarely seen (but usually of lesser degree) in regenerative conditions following hæmorrhage or some toxic agency. The excess of leucocytes fits in with my views on "erythroleukæmia" (cf. Weber and Bode, *Klin. Wochenschrift*, Berlin, 1930, ix, p. 2244). By careful Roentgen-ray examination no evidence of any gastric or duodenal ulcer or of any neoplasm can be detected, and it is possible that the dangerous hæmorrhage may have been from gastric or œsophageal varices. It is further possible that the enlarged liver is slightly cirrhotic, but gastric and œsophageal varices can doubtless occur without hepatic cirrhosis.

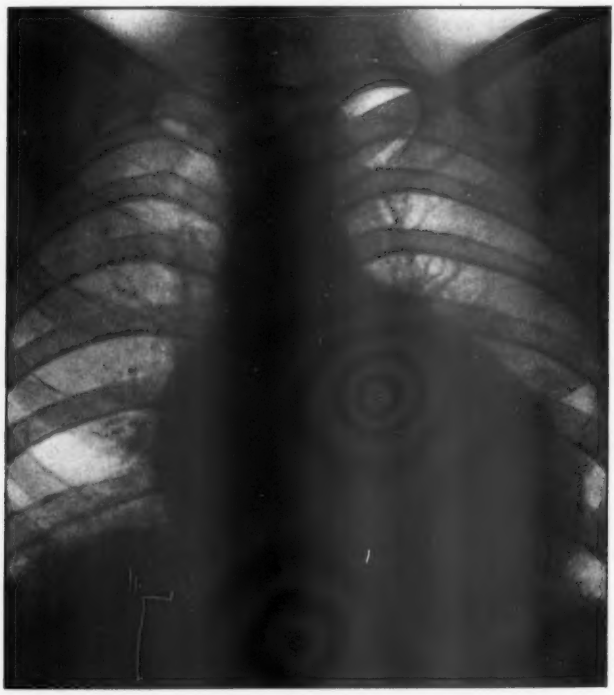
Addendum.—Fractional examination of the gastric contents (October 13, and again October 24, 1931) shows complete achlorhydria, even after a subcutaneous histamin injection; pepsin present.—F. P. W.

Cardiac Malformation with Compensatory Polycythæmia. ? Septal Deficiency.—F. PARKES WEBER, M.D., and M. SCHOLTZ, M.D.

The patient, G. T., aged 23, a furniture-packer, though a strongly built man, accustomed to heavy work, has always had cyanosis, at all events on exposure to cold and on exertion. According to his mother he was a "blue baby" at birth. During the last year he has had occasional attacks of vertigo of short duration, which our aurist, Dr. W. Wilson, thinks may be of aural origin (catarrhal obstruction in one Eustachian tube). Otherwise he has enjoyed good health. By ordinary examination and Roentgen radiograms (see figure, p. 12) the heart is found to be bilaterally equally enlarged to left and right. By auscultation often no definite murmur can be made out, but a musical systolic sound seems to have been heard recently, and sometimes a slight præsystolic murmur and thrill can be detected at the apex, which is in the fifth intercostal space just outside the nipple line. Very slight "clubbing" of fingers and toes. Pulse regular, about 60 per minute. Brachial blood-pressure: 105/90 mm. Hg. No enlargement of liver or spleen. No orthostatic albuminuria. Blood-count on September 7, 1931: Hæmoglobin 145%; erythrocytes 6,700,000; leucocytes 7,400 (eosinophils 4%; polymorphonuclear neutrophils 60%; lymphocytes 32%; monocytes 4%). Later on (Sept. 23) the blood count was: Hæmoglobin 134%; erythrocytes 7,380,000; colour-index = 0·9; leucocytes 6,500. The patient's cyanosis (including conjunctivæ, mouth and pharynx) is accompanied by a turgid appearance, suggesting true plethora in addition to the secondary polycythæmia. The first blood-volume estimation in congenital cardiac cyanosis was by Haldane and Douglas in Weber and Dorner's case (*Lancet*, 1911, i, 150).

The essential cardiac malformation in the present case is almost certainly a septal deficiency, probably of the interventricular septum ("maladie de Roger"), but a gaseous analysis of the arterial blood has not been carried out, which might

prove that a true admixture of arterial and venous blood takes place (always, or at times) within the patient's heart (according to the observations of Campbell, Hunt and Poulton, *Journ. Path. and Bact.*, 1923, xxvi, 234).



Cardiac malformation. Skiagram showing bilateral enlargement of heart.

Two Cases of Advanced Unilateral Pulmonary Tuberculosis successfully treated by Thoracoplasty.[1]—PHILIP ELLMAN, M.D.

(I) C. L., female, single, aged 32, clerk, notified as a case of pulmonary tuberculosis (sputum T.B. +) in 1919; had left-sided pneumothorax induced in 1928 for extensive infiltration of left upper and middle zones of left lung with cavitation of left upper zone. Right lung showed no evidence of tuberculous infiltration either clinically or radiologically. After the pneumothorax, patient had neither cough nor sputum and her general condition had improved; weight then 8 st. Refills continued until February, 1930, when, owing to pleural adhesions, they were discontinued.

Patient soon began to lose weight; a cough with blood-stained expectoration developed. In May, 1930, weight was 7 st. 8 lb. and quantity of sputum expectorated in twenty-four hours increased to 2 oz. It was mucopurulent and blood-stained; tubercle bacilli were present in moderate numbers. Physical signs in left lung were those of extensive cavitation of left upper lobe with evidence of a thickened pleura at base.

X-ray examination of chest (Dr. Stanley Melville): Heart, mediastinum and trachea displaced to left side; diaphragm on left side immobile. Left lung

1 The illustrations for Dr. Ellman's cases (pp. 13-16) are reproduced by kind permission of Messrs. H. K. Lewis.

showed excavation of upper zone and considerable fibrosis and pleural involvement. Right lung showed compensatory emphysema but no infiltration.

Since the lesion was unilateral and showed marked evidence of fibrosis, good collapse of the left lung was essential for any progress. Mr. Tudor Edwards saw the patient for me and performed left thoracoplasty.

Operation.[1]—2.7.30. Left phrenic evulsion. Phrenic nerve found crossing scalenus anticus high up. Length evulsed 1¼ in.

5̄23.7.30.—Upper stage extrapleural paravertebral thoracoplasty. Gas-and-oxygen anæsthetic. Ribs 1 to 5 resected subperiosteally from the transverse process of the vertebræ. Total length evulsed 16 in.

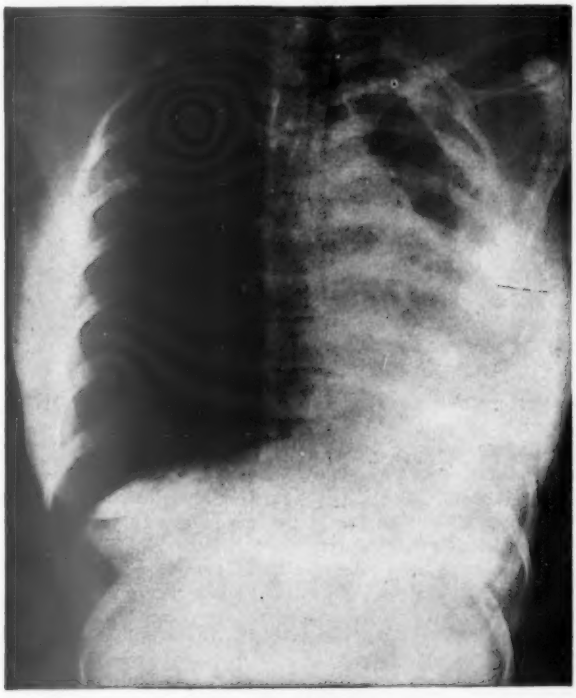


FIG. 1.—Skiagram before thoracoplasty, showing extensive fibrotic disease of the left lung with cavitation of the upper zone. The heart, trachea and mediastinum are drawn over to the left.

6.8.31.—Lower stage extrapleural paravertebral thoracoplasty. Ribs 6 to 10 resected as above. Total length 27 in., i.e., in both stages 43 in. of rib removed.

In September patient went to Frimley for sanatorium treatment until March 23, 1931, when she was discharged. Dr. Wingfield reported that she had made uninterrupted progress at the sanatorium.

She is now, fifteen months after the thoracoplasty, in excellent health; weight 9 st. 3 lb. (gain of 1 st. 3 lb.). No cough; only minute trace of sputum, repeated examinations of which have shown no tubercle bacilli. Slight dyspnœa on exertion due to some compensatory emphysema.

[1] Notes kindly supplied by Mr. Tudor Edwards.

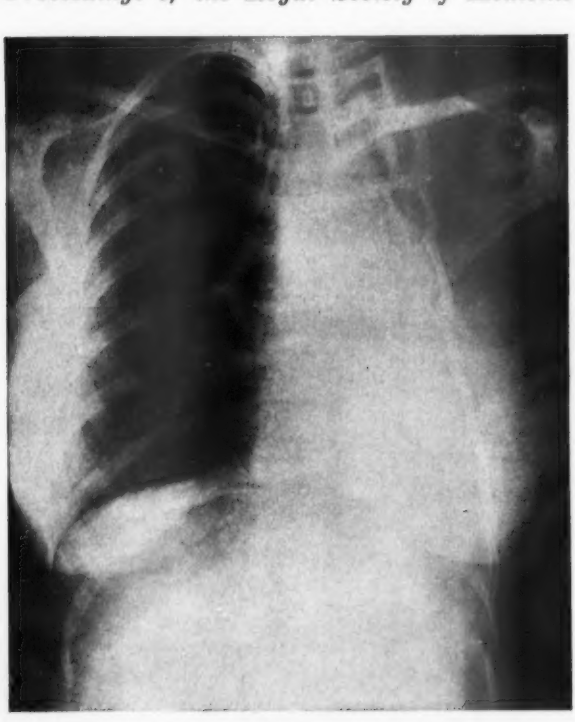


FIG. 2.—Complete thoracoplasty.

(II) Patient, L. L., male, married, aged 28, compositor, first entered sanatorium in June, 1928, having a definite tuberculous infiltration of right upper and middle zones, with cavitation of right upper zone. Sputum: Tubercle bacilli in large numbers; mucopurulent; patient expectorated about 1½ oz. in twenty-four hours. Right artificial pneumothorax was induced some time later and a satisfactory collapse obtained. General condition steadily improved; in August, 1929, he was doing full-time work and having regular monthly refills. Continued well until February, 1930, when he complained of pain in right side and hydropneumothorax soon developed.

Refills were continued for two months, but in April, owing to considerable pleural involvement, they were discontinued. Condition remained fairly satisfactory until August, 1930, when he had a sudden large hæmoptysis (2 pints). Physical signs showed marked re-activity in right lung (large cavity in upper lobe with pleural thickening at base), heart being drawn over to the right side. Left lung: No abnormal physical signs.

X-ray examination (Dr. Stanley Melville) confirmed the clinical findings, and after patient had had one month of absolute rest and his general condition had improved, Mr. Tudor Edwards performed thoracoplasty.

Operation (10.12.30).—Right phrenic evulsion. Horizontal incision. Length evulsed 12 in. Nerve crossed scalenus anticus at normal site.

22.12.30.—Upper stage paravertebral thoracoplasty. The ribs were found to be triangular, lying in a position of forced expiration; ribs 3 and 4 were joined together

by a bar of bone. Length evulsed 18 in. Ribs 1 to 5 resected subperiosteally from the transverse process forwards.

2.1.31.—Lower half paravertebral thoracoplasty. Ribs 6 to 10 resected as above. Length 30 in., i.e., 48 in. in the two stages. Very little reaction for two or three days to each operation.

The thoracoplasty was sufficiently successful to enable the patient to complete the treatment under sanatorium conditions at Frimley from February to June, 1931, since when he has been back at work doing nine hours per day. Now he has neither cough nor sputum and is completely afebrile; general condition very good although he has not, as yet, gained much weight (weight before thoracoplasty, 9 st. 4 lb.; present weight, 9 st. 5½ lb.), but he is doing a full day's work.

"Idiopathic" Spontaneous Pneumothorax with Complete Lung Expansion within One Month.—PHILIP ELLMAN, M.D.

W. J. C., married man, aged 65, a leather cutter (work involving hard manual labour), sent to my clinic early in November, 1930, with the following history:

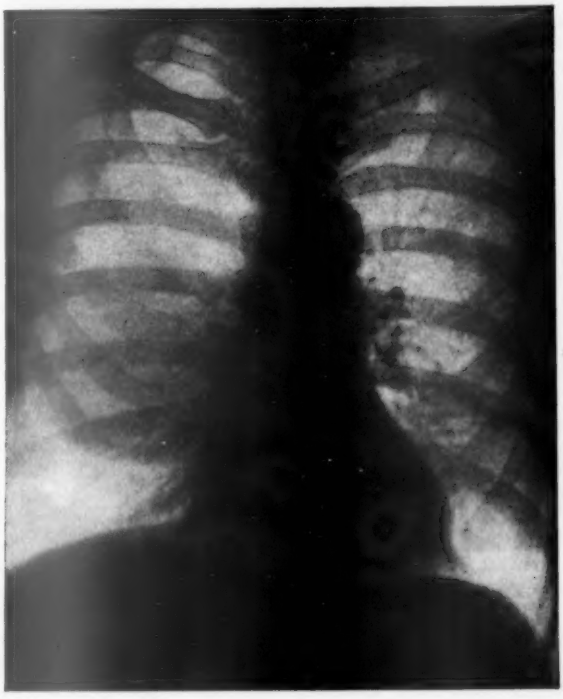


FIG. 1.—Skiagram of the case of idiopathic spontaneous pneumothorax. N.B.—The right lung is collapsed and has the appearance of a ball around the hilum.

Eight days ago, whilst eating a meal, a sudden attack of dyspnœa and severe pain developed in the right side, followed by a coughing attack. His doctor reported that he was cyanosed and suffering from shock. When I saw him he was obviously dyspnœic; no cyanosis; pain in right chest not so severe. Pulse regular, 100; he was afebrile.

Examination of chest.—Right side moved badly; apex-beat well outside normal limits. Whole of the right hemi-thorax was hyper-resonant to percussion, breath sounds markedly diminished, and a definite metallic tingling was heard, especially after coughing. There were no abnormal physical signs over the left lung.

The condition was undoubtedly one of spontaneous pneumothorax; radiological examination by Dr. Mather Cordiner showed that the lung had collapsed like a ball around the hilum.

When the patient was seen fourteen days later, after complete rest, the lung showed definite evidence, both clinically and radiologically, of expansion, and within one month it had completely re-expanded.

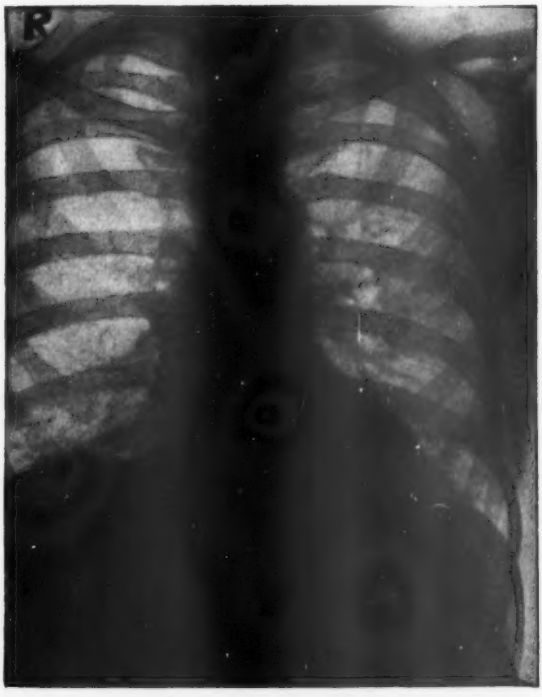


FIG. 2.—Skiagram showing lung completely re-expanded.

When first seen the patient weighed 8 st. 6 lb.; he now weighs 9 st. He has been back at work since February, doing hard manual labour and feels perfectly fit. During his illness, when he had a slight cough and expectoration, repeated sputum examinations were made but tubercle bacilli were never found. There is now neither clinical nor radiological evidence of lung disease.

This case is probably one of the group of cases of simple or "idiopathic" spontaneous pneumothorax occurring in healthy individuals, in which the lung usually re-expands completely in from one to two months, and little further trouble appears to result.

[*The report of other cases shown at this Meeting will be published in the next issue of the* PROCEEDINGS *of the Section.*]

Section of the History of Medicine.

President—Dr. R. O. MOON.

[*October* 7, 1931.]

PRESIDENT'S ADDRESS.

Van Helmont, Chemist, Physician, Philosopher and Mystic.

By R. O. MOON, M.D.

FOR the beginning of our session this year it has seemed to me that it might be profitable to offer you some account of a physician, who may be regarded as the founder of the Iatrochemical School of Medicine and who took for his province almost the whole realm of knowledge.

Van Helmont was not only a physician but a chemist, also a philosopher and mystic. It will, I am afraid, therefore be only in rather a superficial manner that I can in this paper touch on these various aspects of his life's activity.

Born at Brussels in 1578, Van Helmont came of a noble family. In his youth he was characterized by an irresistible thirst for knowledge. He went to the University of Louvain and finished his studies in 1594 at the age of seventeen. While at the University he was much interested by the lectures of Martin del Rio on Magic, but as he found these gave him no real satisfaction, he betook himself to the study of the philosophy of the Stoics, in which he hoped to find inward peace, and he was particularly pleased with the writings of Seneca and Epictetus. But he soon came to the conclusion that Stoicism is but a form of pride, and that man, without the inspiration of God, is a thing of nought and a vain shadow. He was then offered a rich living if he took orders, but he was frightened by the saying of St. Bernard that he would live upon the sins of the people.

In consequence of this he turned aside to more worldly studies and busied himself with law and political science, and thought of becoming a barrister. Next he directed his attention to botany and, as he himself expressed it, "when tired and wearied with too much reading of other things, for the sake of recreation I rolled over Matthiolue and Dioscorides, thinking within myself that nothing was equally necessary for mortal men, as by admiring the grace of God in vegetables to minister to their proper necessities and to crop the fruit of the same. I found the art of Herbalism had nothing increased since the days of Dioscorides."[1]

This study of botany led him to think of taking up medicine. For a long time, however, he hesitated about adopting such a profession, as he feared lest so plebeian an occupation might be a stain upon his illustrious ancestry. But one night in a dream—and throughout his life Van Helmont attached great importance to dreams—he received an inspiration from his guardian angel—Raphael—who apparently took no cognizance of his noble pedigree, and that decided him to take up medicine, which he accordingly did, as he expected that in this profession he would find complete satisfaction alike for his thirst for knowledge and for his love of mankind.

Van Helmont's diligence was amazing, for he simply devoured medical books, both ancient and modern, reading Galen twice and knowing the Aphorisms of Hippocrates by heart. After this, at apparently about the age of twenty-two, he began to give a course of lectures on surgery at Louvain till he felt that his experience was insufficient. But by degrees he became disquieted with what he saw of medical practice. "Then it came into my mind," he says, "that the art of Medicine was full of deceit, without which the Romans had lived happily five hundred years. I reckoned the Greek art of healing to be false. We know well how to dispute about every disease, but we cannot radically cure the pain of toothache or the itch." Finally, the study of devotional and mystical books such as those of Thomas à Kempis, author of the *Imitatio Christi*, and Tauler, the German mystic of the fourteenth century, decided him to abandon medicine altogether.

[1] *Oriatrike*, Translation of J. Chandler.

In consequence of this he left his native land with the intention of never returning. He wandered through Switzerland, Italy, France and England. In 1604 he was in London, where he had intercourse with the leading people of the day, and was introduced to the Queen. But he did not meet with more liberal views abroad; everywhere he found the same sluggishness and ignorance, so in 1605 he decided to return to his native land.

On arriving at Antwerp he found the plague raging and, as he said, everyone forsook the sick, and unfaithful helpers, distrusting their own art, more swiftly fled than the unlearned common people. His sympathy with human suffering decided him to take up medicine again. In 1609 he married Margaret Van Ranst, a rich heiress, and settled down to a life of study and medical practice at Vilvorde, near Brussels, till his death in 1644.

Van Helmont soon made a most determined attack on traditional medicine, and in particular he was opposed to the teaching of Aristotle, Galen and the whole Arabian school of medicine which flourished at that time. About a century previously his famous predecessor Paracelsus had launched a similar attack on Galen and traditional medicine. Paracelsus intended to make all things new and plunged into the wildest extravagances, thereby discrediting much that might have been valuable in his writings. Unlike Paracelsus, Van Helmont was a man with a disciplined mind; he had studied anatomy as well as botany, besides having received what was then regarded as a liberal education.

Van Helmont was keenly opposed to the demonstrations of deductive logic as a means of obtaining scientific truth. "The knowledge," he said, "which we have by demonstration was already in us and is only made a little more distinct by a syllogism: but it remains as before joined with doubting: because every conclusion doth necessarily follow the weaker part of the premises, hence it comes to pass, that it is composed with a doubt of the contrary." "Real knowledge must in its very nature involve intuition, for it to be possible the knower and the thing known must in some way become identified. Every kind of true or intellectual knowledge is not to be demonstrated, that is, true sciences cannot proceed from demonstration." "Logical invention is a mere retaking of that which is known before. Thus what is not known, logic knows not. I could desire the schools to tell me what Science logic has ever brought forth to light? Whether happily Geometry? Music? Making of Glass? Printing? Husbandry? Medicine? or Building? or any profitable science? Verily none?"

After this destructive criticism one naturally asks what contribution did Van Helmont make to medicine, apart from curing numbers of patients in strange and mysterious ways. His main ideas are embodied in his work, *Ortus Medicinæ* which, however, was not published till 1648, four years after his death, being edited by his son, Mercurius Van Helmont.

Of all things chemistry was the most important subject on which he threw fresh light, and he has been called the greatest chemist prior to Lavoisier. His great contribution to chemistry was, of course, the discovery of carbonic acid gas (CO_2), which, oddly enough, was entirely neglected by succeeding chemists until Joseph Black, in the middle of the eighteenth century (1757), rediscovered it, naming it "fixed air." Previously, natural philosophers had regarded all gaseous substances as being mere varieties of air. In choosing the designation "Gas," Van Helmont tells us that he had the "Chaos of the Ancients" in mind. "Gas is a far more subtle or fine thing than a vapour, mist or distilled oylinesses, although as yet it be many times thicker than air." He calls this carbon dioxide, "Gaz Sylvestre"—the "Wild Gas" on account of its apparent incondensability. He observed that it was produced when acetic acid acts upon calcium carbonate. He further noticed its occurrence in mineral waters and in the stomach, and he knew that this gas could extinguish the burning of a candle. He also made sulphur dioxide and nitrous oxide. His further chemical progress in this direction

was hindered by the notion which he frankly held, like Thales, that water was the first substance of all material things. This renders his idea of a gas confused, since it had to be explained that gas was formed of water. He endeavoured to prove that the vapour of water was something wholly different from real air. He was the first to explain that the explosive power of gunpowder was due to the production of gas.

With regard to metals, he clearly realized that they continue to exist throughout a series of chemical metamorphoses. Now this was rather a novel conception, for in his day alchemists believed that when iron was immersed in a solution of blue vitriol, it was transformed into copper. For this Van Helmont substituted the more rational notion that the copper deposited on the iron was formerly present in the solution. He also pointed out that silver is not destroyed when dissolved in nitric acid (aqua fortis), but that it is present in the resulting solution and can be obtained therefrom in its original form by means of copper. He also studied the quantitative side of chemical reactions, which is specially to his credit, since in his day little attention was paid to the balance, to the use of which the science of chemistry owes so much.

One of his most remarkable and valuable pieces of work was the discovery that from a given piece of glass, the exact weight of silica can be obtained that was used in the preparation of it. He regarded air as an element, but believed that it possessed no weight; he also excluded it from his theory of the genesis of material bodies, because he regarded water as the first matter or material cause of all things.

We will now consider Van Helmont's contribution to medicine. Like Paracelsus, he regarded all organic processes as based upon chemical events, and these were presided over by spiritual forces which he called "Archæi." This idea of an archæus, or spiritual force, which was the embodiment of energy, first came from Paracelsus. In health, all the different chemical processes are correctly governed by the archæus. Death is the loss of the archæus, when the natural chemical changes are left entirely to themselves without any restraining influence. Disease is the failure of the archæus to govern aright, and is the result of the entrance into the body of germs bringing about chemical changes which the archæus cannot master. But over and above the archæus, Van Helmont thought that there existed a "sensitive soul" which he called a light. "I do not," he said, "mean a burning, heating light, the cause of the heat of the body, for the heat of the body is merely the product of life, of vital actions, and is not life itself." This "sensitive soul" is mortal, and in man co-exists with the immortal mind—*mens immortalis*. It is as it were the mere husk or shell of the mind, and the latter works through it, so that at the bidding of the mind the soul makes use of the archæus, whether it itself wishes to do so or not. Before the Fall of Adam the archæus obeyed the immortal mind and was directly controlled by it, but at the Fall man received also the sensitive soul and with it death, the immortal mind retiring within the sensitive soul and becoming as it were its kernel. The seat of the sensitive soul he located in the pit of the stomach, since a blow in that region destroys consciousness. Though it is placed in a locality, it is nevertheless not there in a local manner, but from the stomach the soul's influence was diffused throughout the body, as the rays of light are brought from the sun to the earth.

Van Helmont does not tell us precisely what the nature of the archæus was, but he appears to have conceived of it as a kind of æther-like structure. The whole economy of the human body was controlled by a hierarchy of these quasi-spiritual principles, the chief being the archæus of the stomach. With regard to digestion, Van Helmont certainly put forth views which were much in advance of his time. In his day the prime agent in digestion was thought to be heat, and it was envisaged as a process of coction. The solution of the foodstuffs was thought to be brought about in the same way as that in which the housewife prepares soup from meat and vegetables. He points out the impossibility of this, for certain

animals are able to digest substances which by no means of cooking can be reduced to a solution. Moreover, in fever, when the heat of the body is increased, the powers of the digestion, so far from being improved, are definitely impaired. On the other hand, Van Helmont likens the process of digestion to that whereby wine is made from grapes or beer from barley; in fact, he thought he saw in these things a clue to the deepest mysteries of Nature's activities. As Sir Michael Foster says: "Previous writers had caught hold of the phenomena of the fermenting wine vat, as being, though mysterious themselves, illustrative of the still more mysterious phenomena of the living body." "Heat therefore," Van Helmont said, is not the author of digestion, but "there is a certain other vital faculty, which doth truly transchange nourishments and that I have designed by the name of ferment, but in us there are many ferments." He realized that fermentation is a process far more complex and subtle than are the ordinary chemical reactions. He never, however, suspected the presence of ptyalin in the saliva and was quite unacquainted with the functions of the pancreas. By means of ferments the archæi of foods are conquered by the digestive archæus of man and nourishment is transmuted into blood. He remarked that the work of the ferment of the stomach which acts in an acid medium ceases when the chyle reaches the duodenum, where the next stage of digestion is effected by the bile. He seems to have thought that the bile was secreted by the gall-bladder, which he called "a noble bowel." He regards all the chyle as being absorbed by the veins and makes no reference to the lacteals, which had been discovered by Aselli of Cremona in 1622. Van Helmont had a good deal to say about fevers. The current view in his time was that the blood in fevers undergoes putrefaction; this he rejected, and indicated that heat is not the cause of a fever, but one of its symptoms due to the disordered activity of the archæus. The archæus attempts to throw off the fever that is attacking it by rigor and trembling, but not being successful, becomes enraged and thus produces feverish heat. "Fever is the effort of the archæus to get rid of some irritant, just as local inflammation is the reaction of the local archæus to some injury." The intermittent character of certain fevers is due to the fact that the archæus, like a wrestler, pauses to take breath, so that he may be better able to shake off his enemy—the fever. The seat of the fever, he considered, was not in the heart, as the Galenists taught, but in the stomach, small intestine and spleen; the nearer the fever to the pylorus the greater was the danger,

In regard to treatment Van Helmont, unlike most physicians of his time, was strongly opposed to venesection. "A bloody Moloch," he said, "sits President in the chair of Medicine"; he thought that it was useless or even hurtful, as diminishing the mass of vital spirits which work in the blood. On one occasion he wrote, "Unless the Lord shall avert it—the life of mortals will dayly be shortened and at length pass into the Grave in its green eare, through the offence of cutting of a vein and purgings." On the subject of diet he said little except to enjoin moderation. "Let the supreme defence of long life be sobriety; otherwise those things which favour do nourish best and a hungry man will easily concoct those foods which do favour him most."

Though he differed from Paracelsus in many points concerning the origin and nature of disease, he seems to have followed him closely as regards therapeutics. Thus he made use of laudanum, which was probably first introduced into medicine by Paracelsus; he also employed preparations of mercury and antimony, which were denounced by the Galenists, and he highly commended wine. In the treatment of maladies, such as gout and pleurisy, which he thought to be due to an excess of acid, he recommended alkaline substances.

But the most important thing in the matter of all medicines and remedies was to pacify and appease the archæus, regulate its functions, and assist it in overcoming the power of disease. In order to cure a disease it is useless merely to alleviate the symptoms; what is necessary is that something shall be done which will act upon

or influence the archæus. Gout, for instance, arises through the anger of the archæus of the stomach, causing it to disperse the acid digestive fluid into remote places in the body, thereby producing a sickness of the joints. Some medicines, such as spices, act upon the archæus by means of their sweet odours or pleasant tastes. Certain drugs do move the archæus not so much "by cleansing and sequestring impurities as by appeasing his griefs and disturbances and a continual and successive substituting of nourishing ideas. In Words, Herbs and Stones there is great Virtue." On some occasions it seems legitimate to frighten the archæus; thus he tells us that dropsy is not due to disease of the liver, as was formerly supposed, but to the wrath of the archæus presiding over the kidneys, who may be reduced to order by frightening him. This can be accomplished by tying a snake round the patient's waist and applying live toads to the region of the kidneys.[2]

Like Paracelsus, Van Helmont believed that each country produced the special medicine suitable for its own diseases, and therefore that it was unnecessary to import foreign drugs. Doubtless, on this principle he would have rejected quinine, for he declares that it is inconceivable that the merciful Father of mankind should have been less merciful to his European children before the discovery of the Indies than afterwards.

Living at the time he did, it seems strange that Van Helmont should have known nothing of Harvey's great discovery, first published to the world in 1628, some sixteen years before the death of Van Helmont. Yet with the Galenists he still believed that the blood was conveyed from the heart to the various organs of the body by both arteries and veins; he still believed in the passage of the blood through the septum ventriculorum, from the right to the left ventricle, while explaining that the hypothetical vital spirits, always present in the left ventricle, were able to pass in the opposite direction.

He did not understand the true function of respiration, but he rightly rejected the current view which supposed that the function of the inspired air was chiefly that of cooling the extreme heat of the heart.

As a contemporary of Bacon and Descartes, it seems strange that he should have believed that wounds could be healed by the sympathetic ointment of Paracelsus, which was applied not to the wound itself, but to the bloody weapon with which it had been inflicted. Under the general name of magnetism he grouped cures known to him of action at a distance, e.g., the attraction of rubbed amber for pieces of chaff, the phenomena of consonance as exhibited by a couple of violin strings. This power, he says, lies in the hidden man, obscure, or as it were asleep, in his present corrupted state. Its activity is, for this reason, restricted to operating within a man's own body, but the possibility remains (and Van Helmont did not doubt it) of its becoming fully awakened and operative on external objects.[3] This power he calls "magical," and says it is the power which the devil uses for his own ends in the case of witches and their like. But the power is certainly not evil in itself; indeed it is God-given, and, if it may be awakened for evil purposes, no less may it be awakened for good. If man be made in the image of God, then, like God, he ought to be able to act on some things by a mere effort of his Will, that is by his Word alone. Such ideas seem of some interest in view of the results of modern experimental research in the domain of abnormal psychology, especially as concerns the phenomena of telepathy and hypnotism, and the philosophy of the Unconscious generally.

He thought that wisdom was a gift of the supreme power and that one must pray in order to obtain it, and that one must give up the exercise of the will if one wishes to take part in this influence of the divine grace. In all the important circumstances of his life a spirit is said to have appeared to him.

Though Van Helmont was a devoted son of the Roman Catholic Church, he came into conflict with it by his *De magnetica Vulnerum curatione*, in which by insisting on

2 E. T. Withington, *Medical History*, p. 308.
3 J. B. Van Helmont, by H. S. and J. M. Redgrove.

the potency of magnetic virtues, he seemed to explain away, on physical grounds, some of the miracles. "What marvel," he says, "if a theologian knows nothing of that matter! For after the priest and the Levite had gone to Jericho, came the layman, the Samaritan, who had taken from the priests all right of enquiring into natural things. Nature therefore has thenceforth not called the theologians as her interpreters, but has adopted the physicians as her sons." In consequence of all this, through the machinations of his medical enemies, the Holy Inquisition of Spain condemned a number of the propositions contained in his books as heretical, and in 1634 he was imprisoned in the convent of the Franciscans in Brussels. Fortunately he only had to remain there a fortnight and was allowed to serve the remainder of his imprisonment in his own house.

Van Helmont is the embodiment of the strife between belief and knowledge, and he has been called the Faust of the seventeenth century. In himself he united the passionate piety of the orthodox Catholic with the free outlook of the philosophic inquirer into Nature. The opposition between the head and the heart, between the discursive reason and the indefinite longings of the emotions seems never to become permanently reconciled. Even such pure physicists as Newton and Kepler have felt that their systems did not cover the whole of experience; still more is it natural that the physician, who deals with life as well as with organic nature, should accept only too readily perhaps ideas which cannot be explained either by the laws of physics or chemistry. The fact is the approach to the Absolute can never be made wholly by the discursive or syllogistic reason, and this seems to have been for ever made clear by Kant, who showed that our knowledge can only be of phenomena. What then is to be done about the extra-phenomenal world, or the "Ding-an-sich," as Kant called it? Can we have any knowledge of this? Here is the field of the pure mystic. It is true that the German philosopher Schelling had some idea of the intellectual intuition of the Absolute, and Hegel had an elaborate logical system by which one can come in contact with the "Thing in Itself," or Ultimate Reality, but such methods can only be employed by the exceptional intellect; it is more usual for any attempts at such forms of knowledge to be made by direct intuitions of the heart, or indeed by visions, and we know that Helmont had frequent visions throughout his life and laid great stress upon them, whereas the highest type of mystic, such as Plotinus, does not appear to attach such great importance to visions or conditions of ecstasy. In some ways we may speak of the problem as how to deal with the non-rational. To most of us, as time goes on, there appears to be a part of our experience which will not submit itself to the rules of logic and the discursive reason, and to no one is this more obvious than to the practitioner of medicine. It is not surprising therefore that we should find physicians casting around for some doctrine or ideas which will pilot them over these uncharted regions of experience, and when there are non-rational ideas in the air, these are very readily seized upon. It is unfortunately not difficult to fall a victim to such ideas in their entirety, and having found that a rational system will not cover everything to assume that it will cover nothing. Hence there sometimes arises a flight from reason altogether, and men, having become distrustful of the validity of the laws of physics and chemistry, find themselves landed in some hopeless or theosophical hotch-potch of Christian Science which is characteristic neither of Science nor of Christianity. If we are prepared to say with Daremberg, "Nothing good can come from medicine either from the *à priori* method, or from mysticism," then we shall have to condemn a good deal of Van Helmont's writings. On the other hand, fanciful explanations in medicine, as in other things, are often stimulating and fertilizing and are perhaps better than none at all. In all scientific discovery imagination is a most important factor. Though there may be much knowledge which is above or beyond reason, such knowledge should not contradict reason.

Section of Neurology.

President—Dr. A. Stanley Barnes.

Cases shown at the Meeting held at the Hospital for Epilepsy and Paralysis, Maida Vale, London, on Thursday, October 15, 1931.

Cerebral Tumour (? Third Ventricle): Onset with Coma.—Douglas McAlpine, M.D.

N. L., male, aged 21, was brought in an ambulance to the Casualty Department of the Middlesex Hospital at 6 p.m. on September 18, 1931. He was comatose, but extremely restless—trying to get off the couch—and it was necessary to hold him down by force at times. The pupils were widely dilated and inactive to light. The deep reflexes were present and there was a double extensor plantar response. When admitted to the Neurological Ward at 8 p.m. he was still comatose and at times struggled violently. No epileptiform phenomena noted. Pupils equal, dilated and centrally placed; did not react to strong light. Fundi normal. No weakness of face or limbs noted. Deep reflexes present and equal. Temperature 102·2. Pulse 136. Respiration 44. Abdominal reflexes absent, all segments. Plantar responses extensor. No rigidity of neck. Heart sounds normal; no enlargement of heart. Blood-pressure 115/60. Bladder moderately distended. A catheter specimen contained no albumin or sugar. Lumbar puncture showed cerebrospinal fluid under pressure (210 mm. of water). No increase in protein; lymphocytes 4 per c.mm.; Wassermann reaction subsequently negative.

When seen again at 11.45 p.m. his condition was the same. Temperature 103·8. Pulse 124. Respiration 32. No rigidity of the neck muscles. Plantar responses still extensor.

September 19: 1.30 a.m.—Regained consciousness for a short time, but relapsed again into coma. 3.30 a.m.—Temperature 99·6. Pulse 106. Respiration 26. Became conscious and vomited; seemed quite sensible. 9.30 a.m.—Was perfectly conscious; stated he had no headache, but complained of double vision. No aphasia. Dilated pupils, equal and inactive to light, somewhat sluggish on accommodation. Ocular movements apparently full; diplopia on looking upwards (disappeared one hour later). Fundi normal. Visual fields full (confrontation and perimeter). No facial weakness. Abdominal reflexes diminished right side. Plantar responses flexor. No neck rigidity.

History.—Is employed in domestic service. Since the age of twelve he has been subject to occasional headaches. During the past three years these have become more frequent and have occasionally been accompanied by vomiting. The headaches are frontal; when they are severe he becomes giddy ("things go round and the floor seems to come up towards me") and he has to support himself. His vision also becomes blurred. About March, 1931, during a severe headache he saw "several things when there was only one." Since then the headaches have been nearly constant. He has never suffered from fits. On the day previous to admission he had a moderately severe headache. At about 3.30 p.m. he remarked to the cook: "I do feel funny, I can see about six of you." Later vision became blurred and he has no further recollection of what happened. Inquiry from the cook showed that by 4.30 he was unable to walk or speak and was so violent that two men were required to hold him down.

September 20.—Vomited several times in the afternoon. Pupils not so large, light reaction to light, brisk on accommodation. No new signs.

September 21.—Reaction of pupils to light brisker. Abdominal reflexes still sluggish on right side. Fundi normal.

Subsequent history.—Has complained of headaches and on two occasions has vomited in the morning. Pupils now react quite briskly to light. No new signs have appeared.

Skiagram of skull.—No definite evidence of increased intracranial pressure. Diploic channels unusually distinct.

Remarks.—The history is suggestive of an intracranial tumour. The attack of coma, preceded by diplopia, with dilated inactive pupils and extensor plantar responses, (unaccountable for by the degree of coma present), persistence of the inactive pupils to light with diplopia on regaining consciousness, and the pyrexia, could be caused by a tumour inside the third ventricle.

Cystic Hæmangioma of the Cerebellum. Operation with Removal of Mural Nodule.—DOUGLAS MCALPINE, M.D., and HUGH CAIRNS, F.R.C.S.

Patient, Mrs. M. T., aged 48.

History.—During August, 1930, she began to suffer from giddiness and frontal headache, chiefly in the morning. These symptoms continued, but she was able to do household duties as usual until February, 1931, when she contracted "influenza" (fever, delirium and pains in the limbs), which lasted about one week. On getting up she walked "as if drunk" and on two occasions she fell. The headaches became more severe and more constant and were accompanied by vomiting. At times she was seized with a severe pain at the back of the neck, which caused the head to be thrown backwards. Her relations now noticed a change in her disposition; she became irritable and bad-tempered. Her memory was frequently at fault; five minutes after a meal she would complain that she had had nothing to eat all day. During April she complained of a "fog in front of the eyes"; on one occasion she saw double. Besides her uncertain gait, it was noticed that she had difficulty in feeding herself, owing to unsteadiness of the right hand. On two occasions she complained of a "pins-and-needles" sensation in the limbs (more especially in the arms). Three or four weeks before admission her speech became "stammering," slow and difficult to understand, but there had been nc misuse of words. During the same period she became increasingly drowsy, and on two occasions she was incontinent of urine.

May 28, 1931.—Admitted to the Hospital for Epilepsy and Paralysis, Maida Vale.

On examination.—Markedly drowsy and could only be roused with difficulty. Confused and partially disorientated. Comprehension and concentration impaired. No aphasia, but speech dysarthric. A proper clinical examination was only possible after magnesium sulphate had been given per rectum. Temperature 98°. Pulse 60. Respirations 16.

Cranial Nerves.—No anosmia. Pupils circular and equal and reacted somewhat sluggishly to light. Ocular movements full. No nystagmus (later an inconstant slow nystagmus to the right was noted). Bilateral papillœdema; left 5D, right 4D of swelling, with numerous hæmorrhages. No retinal angioma. Visual fields full (confrontation). Left corneal reflex diminished. Right lower facial weakness variable in degree. No deafness. Remaining cranial nerves intact. Head inclined towards right shoulder; at other times retracted.

Upper and lower limbs.—Slight diminution in power in right arm, otherwise power normal. Ataxia and dysmetria present all four limbs, especially right upper. Generalized hypotonia. Reflexes present and equal on the two sides. Plantar responses flexor. Tactile and pain sensibility intact. No astereognosis. Postural sense impossible to test owing to lack of concentration.

Skiagram of skull.—No positive information except signs of increased intracranial tension.

Diagnosis.—A tumour of the right lobe of the cerebellum was suggested by (1) early history of giddiness, subsequent development of ataxia and occipital pain, (2) physical signs.

Operation. June 2.—Local anæsthetic. Both lateral ventricles tapped and found to be dilated. Cystic cavity, size of tangerine, occupied depths of right cerebellar lobe. From upper and inner surface projected a nodule the size of a walnut. It was soft, bled freely and contained some cysts. It was but loosely attached to the surrounding brain and was removed without much difficulty.

Histological examination of tumour.—Typical hæmangioma.

Subsequent history.—For forty-eight hours following the operation there was an exaggeration of the previous mental symptoms with insomnia.

June 5.—Mental condition now nearly normal. General condition very satisfactory.

June 6.—She asked for *The Times* and her needlework.

June 8.—Not so well. Temperature and pulse dropped, suggesting recurrence of hydrocephalus. A large amount of cerebrospinal fluid withdrawn by lumbar puncture.

June 10.—50 c.c. cerebrospinal fluid withdrawn. Considerable improvement.

She subsequently made an uninterrupted recovery and was discharged to a convalescent home on June 30. She walked steadily from the first. Mentally she was quite clear and quite a different woman. Still 2D swelling right disc; 3-5D left. Below the left disc was a subhyaloid hæmorrhage. $\frac{6}{12}$ vision both eyes. Very slight nystagmus on looking to right. Slight weakness right lower face. Speech slightly dysarthric. Finger-nose test showed slightest degree of ataxia.

October 2, 1931.—Improvement in every way continues. Still complains of giddiness at times, especially on turning sharply; is able to do her housework.

Disc edges indistinct, but no papillœdema. Vision—both $\frac{6}{6}$. The subhyaloid hæmorrhage still persists below left disc. The slightest degree of ataxia right upper limb remains. Gait normal.

Left Frontal Meningioma.—HUGH CAIRNS, F.R.C.S.

Mrs. J. W., aged 56, admitted to the Hospital for Paralysis and Epilepsy, Maida Vale, under the care of Dr. Douglas McAlpine, on May 15, 1931.

History.—All her life she had had occasional headaches. In 1927, after the death of her husband she became depressed and apathetic, whereas formerly she had been a lively and humorous person. In 1929 she began to have bouts of severe headache. In September, 1930, after visiting a cinema, her sight was for a few minutes obscured by rings of light. Attacks of this nature continued and were usually associated with headache; in some of them vision was completely lost. From October, 1930, onwards, she was unable to read even with glasses. A diagnosis of hypertensive retinitis was made. She became rather sleepy and very forgetful, worried and irritable, and she lost weight. From November, 1930, onwards, headaches occurred daily in the left frontal and temporal regions, but they were never violent and there was no vomiting. She was unusually thirsty, and began to complain of buzzing in the left ear.

On examination.—A stout, drowsy woman, unable to give her history. Memory, comprehension and concentration greatly affected. At times she had a little difficulty in naming objects and showed slight perseveration, but her speech was otherwise normal. Sense of smell diminished at left nostril. Bilateral papillœdema (right 5 D., left, 2 D.). Visual acuity: right $\frac{3}{60}$, left $\frac{3}{18}$. Nystagmoid movements on looking to right. Slight left ptosis and diminution of upward movement of left eye. Slight lower facial weakness on right side. Tremor of both upper limbs, more on right side. Absence of abdominal reflexes, slight increase of right knee-jerk, doubtful extensor response on left side. X-rays showed signs of increased intracranial pressure and slight erosion of the outer part of the left sphenoidal ridge.

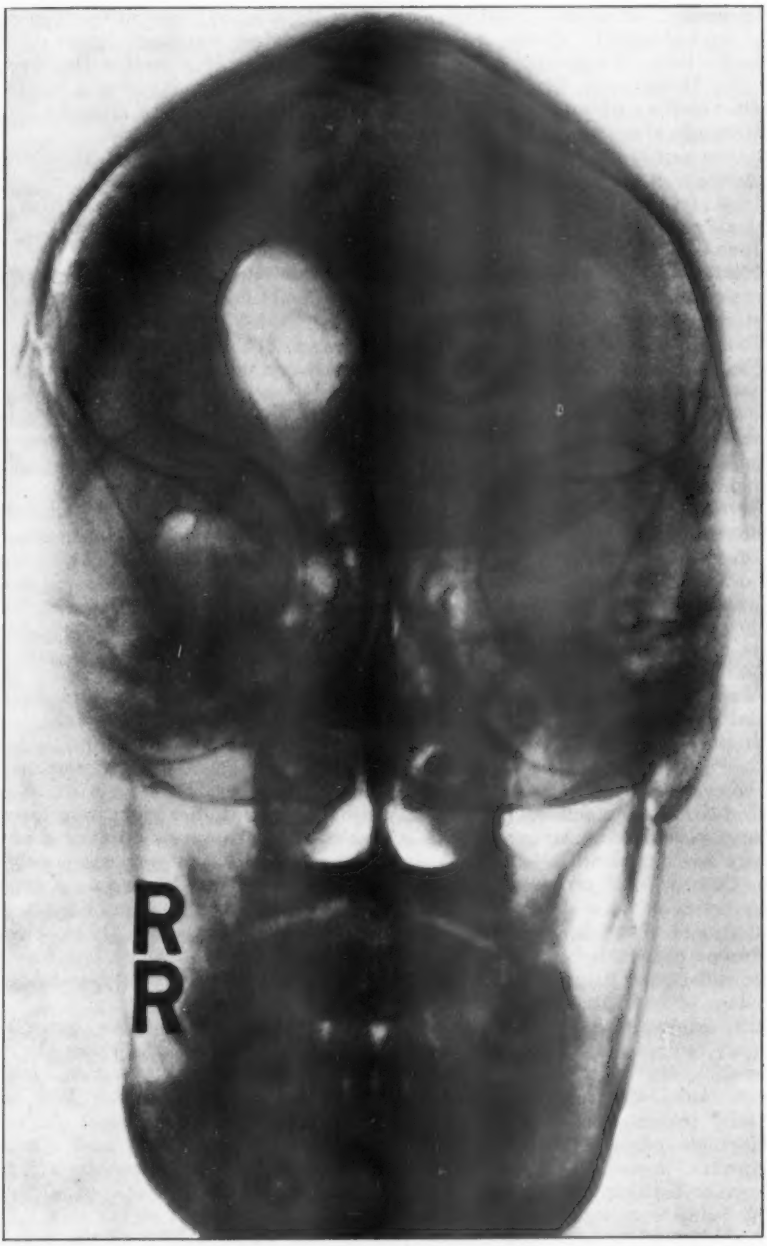


FIG. 1.—Left frontal meningioma. Air in the right ventricle, which is displaced to the right. No air has passed to the left ventricle.

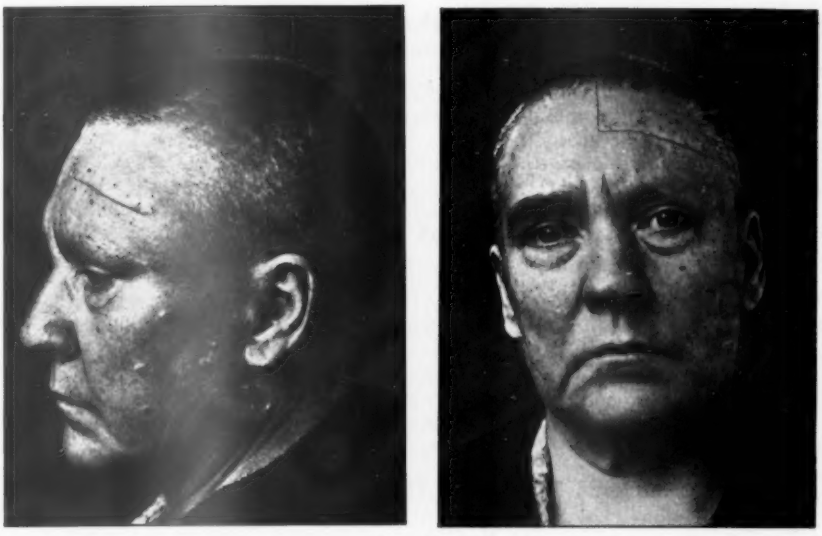


FIG. 2.—Left frontal meningioma. Left frontal osteoplastic operation: the incision fifteen days afterwards.

FIG. 3.—Meningioma of left sphenoidal ridge after removal.

Diagnosis.—It was considered that the most likely diagnosis was one of meningioma of the left sphenoidal ridge, but that ventriculography should be done to confirm this opinion.

Ventriculography (June 16, 1931).—35 c.c. + of fluid in the right ventricle under a pressure of only 50 mm. of cerebrospinal fluid. The right lateral and third ventricles were displaced to the right. No air passed to the left side (fig. 1). There was evidently blockage of the left foramen of Munro.

Operation (June 16, 1931).—Intratracheal gas-and-oxygen. A left frontal osteoplastic exploration (fig. 2) showed a meningioma weighing 60 grm. attached to the dura on the roof of the left orbit and left sphenoidal ridge. The tumour was completely removed (fig. 3).

Histological examination showed a meningioma.

Subsequent course.—For a few days after operation speech was practically limited to "Yes" and "No," the right facial weakness was more pronounced than before operation, and there was weakness of the right upper limb. These symptoms had almost disappeared when the patient was discharged from hospital, twenty-four days after operation, and there was very great improvement in her mentality.

Two months after operation she was quite free from symptoms and had resumed her old activities. Her mentality was normal. Visual acuity: right $\frac{3}{36}$, left $\frac{6}{9}$.

Pituitary Adenoma Treated by Operation.—HUGH CAIRNS, F.R.C.S.

John R., tailor, aged 41, admitted to the Hospital for Paralysis and Epilepsy, under the care of Dr. Douglas McAlpine, on May 22, 1931.

History.—Nine weeks before, his sight became misty, and he found he could not see traffic approaching him from the left side. The trouble began in the left eye but subsequently became worse in the right. He could not see to thread a needle and then became almost blind. In addition he complained of constantly feeling cold, slight frontal headache on one occasion, occasional giddiness on movement, and for

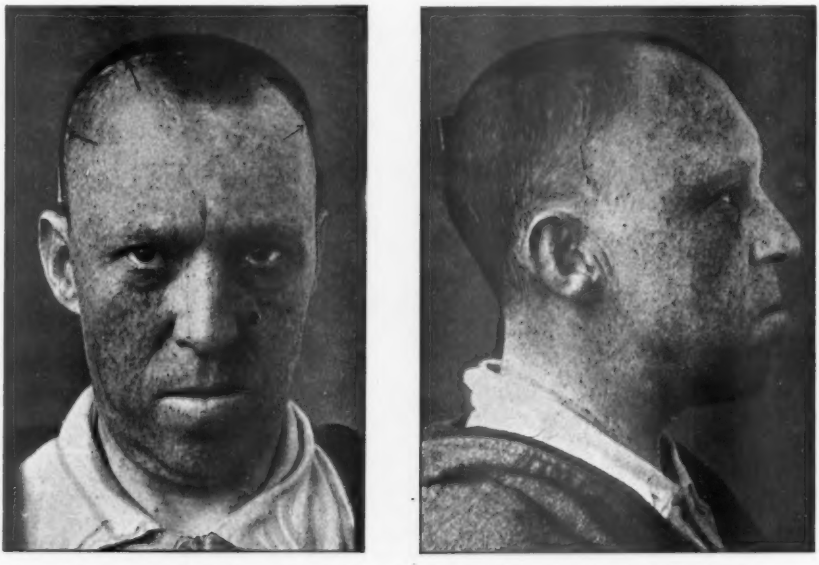


FIG. 1.—Pituitary adenoma treated by operation. Skin incision for right transfrontal exploration; thirteen days after operation.

three months before admission had had failure of libido. He became sleepy and depressed. His wife said he was just like a child in his relish for sweets, particularly in the month before admission.

On examination.—Below medium height and inclined to stoutness. His beard was scanty and it appeared that he had never needed to shave more than twice a week. The pubic hair was of female type and elsewhere his trunk was hairless. The supraorbital ridges were a little prominent and the jaws slightly prognathous, but the hands were not enlarged.

The optic discs showed slight primary optic atrophy. Visual acuity: right $\frac{1}{60}$, left $\frac{6}{18}$. Vision in the right eye seemed limited to the lower nasal quadrant; complete temporal hemianopia of the left eye. The sella turcica was enlarged and shallow. Fluid intake 50 oz., output 50 oz., in twenty-four hours. Fasting blood-sugar, 0·095%.

Operation.—June 9, 1931. Intratracheal gas-and-oxygen. Right transfrontal exploration (fig. 1). The tumour was bulging up tightly in front of the post-fixed optic chiasm. It was freely removed and the tension on the optic chiasm and nerves was thus completely relieved.

Subsequent course.—For the first two weeks after operation there was increased thirst (up to 142 oz. fluid intake per day), and fluid output was as high as 160 oz. per day. This gradually disappeared without treatment. Vision rapidly improved, so that before discharge from hospital fourteen days after operation patient was able to thread a needle. One week later visual acuity was $\frac{6}{12}$ in right eye and $\frac{6}{6}$ in left eye. The fields were almost as full on the temporal as on the nasal side, but there was marked central contraction, as had been observed in the field for the left eye before operation, and it was thus impossible to measure accurately the process of recovery.

Acoustic Neurinoma of Right Cerebello-pontine Angle. Complete Removal. Spontaneous Recovery from Post-operative Facial Palsy.—HUGH CAIRNS, F.R.C.S.

Mrs. E. B., aged 47, admitted to the Hospital for Paralysis and Epilepsy, Maida Vale, under the care of Dr. Douglas McAlpine, on April 2, 1931.

History.—In 1926 she was in bed for several weeks on account of weakness and shortness of breath. She returned to work, but from that time onwards suffered from attacks of vertical headache. Twelve months before admission the headaches became worse and her sight and hearing began to fail. She became unsteady in walking, and drowsy, and began to have attacks of vertigo. Two months before admission she gave up work. For years she had suffered from noises in the ears ("like engines"), and had at times seen double. Vomiting occurred for the first time in April, 1931, after lumbar puncture.

On examination.—The patient was somewhat irritable, and her memory was poor, but her mentality was not otherwise affected. Slight dysarthria, pain on extreme flexion or rotation of the neck. Bilateral papillœdema (right 6 D., left 5 D.) with numerous large hæmorrhages, for the most part pre-retinal, and folds of bright yellow exudate spreading from each disc towards the macula. Visual acuity, $\frac{6}{18}$ in each eye. Great enlargement of blind spots. Nystagmus on lateral movement of the eyes, especially to the right; rotatory nystagmus on looking upwards. Great diminution of right corneal reflex. Slight right facial weakness (all segments). Loss of taste on right side of tongue anteriorly. Hearing completely lost at right ear, normal at left ear. Caloric test.—No response at right ear, normal response at left. Slight weakness of right sterno-mastoid and trapezius. Tremor and ataxia of all four limbs, more marked on right side. She walked unsteadily on a wide base, and when standing tended to fall backwards and to the right. Blood-pressure 190/130. Skiagram of the skull showed signs of raised intracranial pressure. The cerebrospinal fluid, obtained by lumbar puncture, contained 0·7 per cent. protein and 2 cells per c.mm.

Operation.—April 16, 1931. Intratracheal gas-and-oxygen. The cerebellum was exposed by an extensive osteoplastic flap of the type described by de Martel. The left lateral ventricle was tapped, and was found to contain a large amount of cerebrospinal fluid under pressure of 250 mm. cerebrospinal fluid. This pressure-reading was made after the bone flap had been broken back, but before the dura was opened. The cerebellar tonsils were displaced through the foramen magnum to the upper border of the atlas. In the right cerebello-pontine angle there was a tumour, approximately 4 cm. in diameter. It was embedded in the anterior surface of the right lateral lobe of the cerebellum, and the arachnoid cistern covering its free surface contained a layer of opaque yellow fibrinous material. The tumour was circumscribed

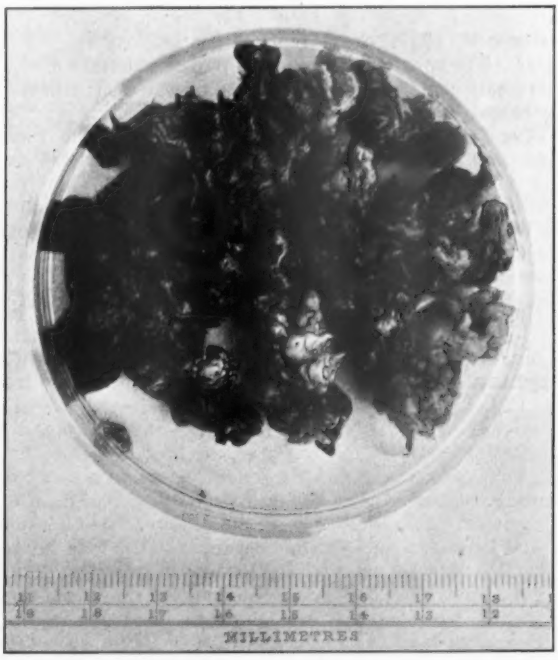


FIG. 1.—Acoustic neurinoma. Fragments of acoustic tumour after removal. A considerable part of the tumour was removed by suction.

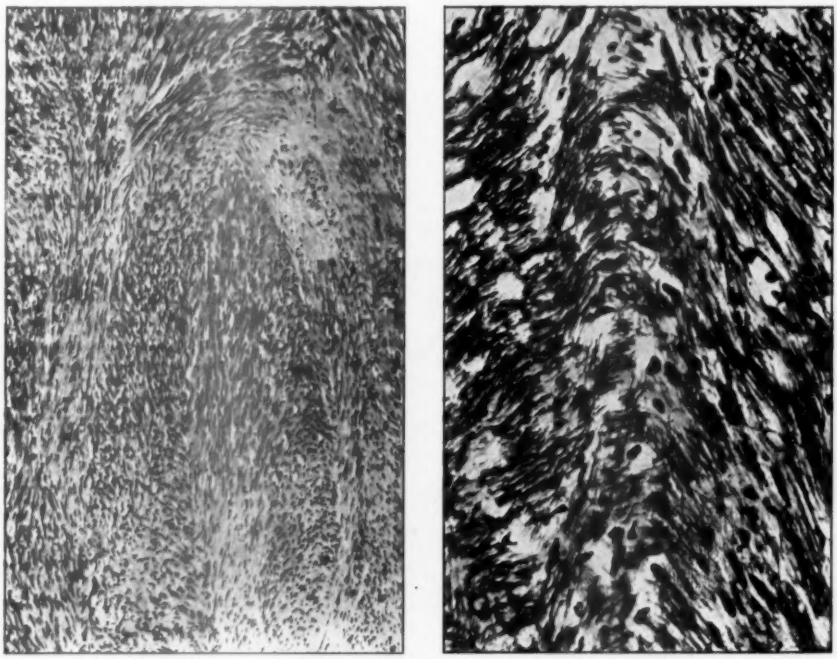
and of nodular outline, and consisted of greyish white tissue and, in the centre, areas of bright yellow tissue. Its capsule was unusually thin.

The tumour and its capsule were removed completely in pieces (fig. 1). Under direct vision it was dissected away from the ninth, tenth and eleventh cranial nerves, which were lightly adherent to its lower surface. After the main part of the tumour had been removed the thinned seventh and eighth cranial nerves were seen on the upper surface of the tumour. They were stripped off the medial part of the tumour, but were partially divided with a vessel, probably the internal auditory artery, at the region of the internal auditory meatus before they had been properly identified in this region. The tumour did not extend into the internal auditory

meatus. The fifth cranial nerve was not seen during the operation. 500 c.c. of citrated blood were given during the concluding stages of the operation.

Histological examination.—Acoustic neurinoma (fig. 2).

Subsequent course.—Immediately after operation there was persistent rapid coarse nystagmus towards the left and a right facial palsy that was complete except that the right eye closed when the patient made grimaces. Her dysarthria was much worse and two days after operation she became completely aphonic and remained voiceless for three days. She was very irritable and at times restless, and was incontinent of urine. For a few days the abdominal reflexes, and knee- and ankle-jerks were lost. On the fourth day after operation herpes appeared on the right side



(*a*) × 85 (*b*) × 300

FIG. 2.—Acoustic neurinoma. (*a*) Stained hæmatoxylin and eosin. (*b*) Laidlaw's stain.

of both lips. She got up on the 12th day. On the 18th day she had a sudden pain in the right side of the chest, which was aggravated by breathing and was associated with fever and tachycardia (? a small pulmonary infarct). She recovered from this in four days.

One month after operation her mental condition was normal She was still very dysarthric. Papilloedema had partially subsided with preservation of vision (right, $\frac{6}{18}$, left, $\frac{6}{12}$). There was nystagmus, more to the right, diminished right corneal reflex, almost complete facial palsy and complete deafness on the right side, slight weakness of the right sterno-mastoid and trapezius, weakness, ataxia and hypotonia of the right arm and leg. She could not walk without assistance and tended to fall

to the right. The reflexes were all present and normal, except that the abdominal reflexes on the left side were diminished and there was still occasional incontinence of urine. On discharge to Convalescent Home, six weeks after operation, she could walk a little with the aid of a stick but was still very unsteady. Her voice was still very dysarthric. The right facial palsy was still complete, except in the orbicularis palpebrarum, and taste was still lost on the right side of the tongue anteriorly.

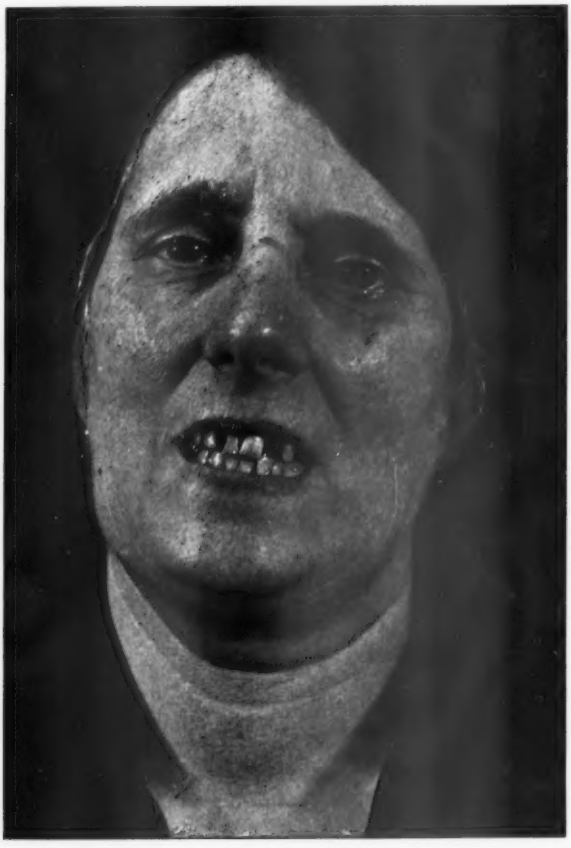


FIG. 3.—Photograph of patient six months after operation showing recovery from right facial palsy.

She was quite free from headaches, tinnitus, and from attacks of vertigo. There was now no incontinence of urine.

Condition 4½ months after operation.—Her walking was greatly improved: she went about the house by herself doing her own work, but only went out of doors on the arm of her daughter. No headaches. Papilloedema had subsided. Visual acuity: right $\frac{6}{9}$, left $\frac{6}{6}$. Nystagmus on lateral deviation of the eyes persisted. Slight diminution of right corneal reflex. Only slight weakness of the right side of the face

(fig. 3). Complete deafness on the right side and no response to cold caloric test on the right side. She still had some ataxia of the right arm and leg and walked on a wide base.

Comment.—When this patient first came under observation papillœdema was intense and it seemed that nothing short of complete relief from hydrocephalus could prevent her from going blind. Therefore, though she was a poor subject for such a procedure, an extensive cerebellar exposure, resembling that described by de Martel [1], was undertaken with the object of removing the acoustic tumour completely. This was accomplished without great difficulty, and the condition of the patient to-night, six months after operation, shows clearly that the result has been satisfactory. Papillœdema has subsided and her visual acuity is almost normal. Her only disability is unsteadiness in walking and this is still improving.

I have shown this case to-night because of the spontaneous recovery from facial palsy that followed removal of the tumour. The right facial and auditory nerves were identified at operation, but it was thought at the time that they were irreparably damaged when the auditory branch of the basilar artery was clipped near the internal auditory meatus. The damage was evidently limited to the more superficially placed auditory nerve, for immediately after operation the patient could still close her right eye, and between the sixth and thirteenth weeks after operation voluntary power returned in the lower part of the right side of the face. There is now only a trace of right facial weakness (fig. 3). Since the thirteenth week after operation appreciation of taste has returned in the right side of the tongue anteriorly.

Two further tumours of the cerebello-pontine angle have been operated upon since this experience, and in each case an attempt was made to preserve the facial nerve. In the first, a meningioma, the attempt failed completely. The facial nerve was running through the substance of the tumour (or was possibly surrounded by lobules of the tumour) and it could not possibly have been saved. At an early stage of the process of piecemeal removal of the tumour this nerve became a great hindrance. I divided it, but finally had to abandon the operation owing to hæmorrhage before the whole of the tumour had been removed. There has been a fairly good result as regards improvement in walking, but the case shows clearly that it will not be possible to spare the facial nerve in all cases.

My second case was a dysarthric, rather cyanosed man, with a large right-sided acoustic tumour, which was unlike Mrs. E. B.'s tumour, for it had a thick capsule (about 0·1 cm. wide), and contained a fairly large blood-clot in its lower part. However, in this case also, the tumour did not project into the internal auditory meatus, and we were able to remove it completely. The facial and auditory nerves were lying above and in front of the tumour and only appeared when the last portion of the capsule was being drawn out. The capsule was divided on either side of the nerves, which were thus left unbroken. Immediately after operation there was complete facial palsy, but there was immediate improvement in conjugate lateral movement of the eyes to the right, which had been almost completely paralysed before operation. We anticipated a result like that in Mrs. E. B.'s case, but nine days after operation this patient developed *B. coli* meningitis, and subsequently *B. coli* cystitis. He died twenty-seven days after operation, and necropsy showed meningitis, ascending pyelitis, broncho-pneumonia and syphilitic aortitis. (The patient had had just before operation a positive Wassermann reaction in blood and cerebrospinal fluid.) At necropsy the facial and auditory nerves were found to be intact, but were covered by a thin layer of clot in the distal half of their intracranial course.

It is doubtful whether the facial nerve could be spared in complete removal of the tumours that project into the internal auditory meatus. About a year ago,

when operating for trigeminal neuralgia by the posterior approach, I encountered a small acoustic tumour lying partly in the meatus. The facial nerve was spared, but I doubt whether removal of the tumour was complete. There appeared to be no justification in this case to curette the internal auditory meatus.

These experiences suggest that it may be possible in a certain number of cases to remove an acoustic tumour and its capsule completely without destroying the facial nerve, a procedure that has obvious advantages over spino-facial or hypoglossal-facial anastomosis. It appears that the facial nerve may be considerably damaged at operation and yet recover if it is left in continuity. In such cases it is advisable to wait some time before doing a spino-facial or hypoglossal-facial anastomosis.

In describing his operation for complete removal of acoustic tumours, Dandy [2] suggested that it might be possible in future to save the facial nerve, for in one of his cases the nerve was identified during removal of the tumour, but was finally inadvertently torn. Among the cases reported by Stewart and Holmes [3] in 1904 there is one (Case 19) operated on by Horsley, in which the facial nerve was evidently saved. The tumour is described as a fibroma, about 3·5 cm. in diameter, which was removed from the left cerebello-pontine angle, where it had been lying under the cerebellum near the seventh and eighth nerves. After operation there was increase of the weakness of the left sixth and seventh nerves. Seven months after operation there was coarse nystagmus to the left, but "no other disturbance of cranial nerve function."

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[1] DE MARTEL, TH., *Surg., Gyn. and Obst.*, 1931, liii, 381. [2] DANDY, W. E., *ibid.*, 1925, xl, 129. [3] STEWART, T. G., and HOLMES, G., *Brain*, 1904, xxvii, 585.

? Multiple Tuberculomata. Case for Diagnosis.—W. RUSSELL BRAIN, D.M.

S. H., male, pantry hand, aged 18.

Previous history.—Pleurisy in 1929. Family history unimportant.

History of present illness.—January, 1931. While at sea suddenly developed weakness of right arm. Three months later right leg gradually became weak.

June, 1931. Unable to use words correctly; developed diplopia; no headache or vomiting.

August 20, 1931.—Admitted to Maida Vale Hospital.

Condition on admission.—Nominal dysphasia; no dyspraxia.

Fundi normal; vision $\frac{6}{6}$ both eyes; right homonymous inferior quadrantanopia; pupils somewhat dilated; react to light but not to accommodation; associated with reaction to light is slight rotation of globe, right counter-clockwise, left clockwise; external ophthalmoplegia, complete left, almost complete right.

Upper motor neurone weakness of face and tongue both sides; spastic right hemiplegia, right upper limb in extension; spasticity and weakness left side; both plantars extensor. All forms of sensibility impaired on right half of body. Incontinence of urine.

Skiagrams:—Skull shows no abnormality. Chest shows slight increase of hilar shadows towards right base.

Blood: Wassermann reaction negative. Blood-count.—Slight erythræmia; R.B.C.s 6,480,000; C.I. 0·83; white count normal.

Cerebrospinal fluid: Pressure 175 mm. of fluid; normal response to Queckenstedt's test; Wassermann reaction negative; protein 0·02%; cells 2-3 per c.mm.; Lange negative.

Has steadily deteriorated since admission. Dysphasia worse; marked dysarthria. Weakness of left side increasing. Attacks of tachycardia and sweating.

Homonymous Hemianopia. ? Posterior Cerebral Thrombosis.—W. RUSSELL BRAIN, D.M.

Mrs. E. S., aged 25.

Previous health good, except for occasional headaches. Family history unimportant.

Four weeks ago developed severe headache and vomited. Within a few hours right side of body became numb and weak and she found she could not see to the right side. No speech disturbance. No loss of consciousness.

On examination (four days later).—Fundi normal. Right homonymous hemianopia, sparing macula. Vision $\frac{6}{12}$ right and left. Pupils equal, right reacted sluggishly to light, left more briskly. Slight right hemiparesis with exaggeration of tendon reflexes, diminution of abdominal reflexes and a doubtful extensor plantar response on the right side. Right relative hemianalgesia, with postural loss.

Radiograms of skull show no abnormality. Blood: Wassermann reaction negative. Blood-pressure 150/90; no cardio-vascular hypertrophy. No valvular lesion. Cerebrospinal fluid: Wassermann reaction negative. Protein 0·01%. Cells 2 per c.mm. Lange negative.

There has been steady improvement in power and sensibility, but hemianopia remains unchanged.

Spinal Tumour (?).—ANTHONY FEILING, M.D.

L. M., female, aged 49, complained of cold patches in legs and stiffness in walking.

History.—Eighteen months ago, sudden sensation of cold running up right leg to knee. Little later same sensation in left leg. Soon after both legs felt heavy and stiff, R. > L. For eighteen months numbness and coldness, pins and needles in both legs; nine months: pain over right lumbar and right buttock; becoming worse.

On examination.—Pupils and discs normal. Cranial nerves normal. Arms normal. Lower limbs: spastic paraplegia. Bilateral ankle clonus present. Abdominal reflexes $\frac{+ \mid 0}{0 \mid 0}$.

Sensation.—Band of hyperæsthesia of about 1 to 2 in. wide over front of abdomen, just above umbilicus. General diminution to touch, pain and temperature below this level. Spine: No appreciable disease.

Blood: Wassermann reaction negative.

Cerebrospinal fluid: Wassermann reaction negative. Total protein 0·03%. Cells fewer than 1 per c.mm. Lange 010. Queckenstedt: initial pressure 100 mm., rapid rise to 200 mm. and rapid fall.

20.9.31.—Skiagram of spine, normal.

Blood-count.—Hb. 80%; R.B.C.s 4,870,000; C.I. 0·82; W.B.C.s 3,800. *Differential.*—Polys. 50%; lymphos. 44%; hyaline 6%.

23.9.31.—Lipiodol injection. Some held up at D.7 vertebra; rest passes to bottom of spine. A week later skiagram still shows some held up at D.7.

Calcification of Spinal Ligaments ? with Nervous Symptoms.—ANTHONY FEILING, M.D.

E. G., male, aged 55.

History.—Five years—pain across lower part of back, pains in calves and "pins-and-needles" in the soles. Symptoms have been growing progressively worse. Walking causes much pain in calves. Eighteen months—throbbing in the ears "like an escape of steam in the left ear." Deaf in left ear over same period.

Palpitation occurs readily; dyspnœa on exertion. Discharged from the Army 1917, because of disease of the mitral valve. Worries over himself a good deal; has had "periods of depression," suffers from insomnia. Smokes 2 oz. of tobacco weekly; occasional alcohol—used to average one or two pints of beer a week.

Previous history.—Gonorrhœa thirty years ago. Bilateral synovitis of knees thirty years ago, in bed three months (? rheumatic fever). Pneumonia nine years ago.

On examination.—Memory and speech normal. Pupils normal. Optic discs normal. Bilateral deafness L. > R. Upper limbs normal. Lower limbs—reduction in power of both limbs, especially flexion of hips and plantar flexion of feet. These movements are painful. Tenderness and hyperæsthesia over calves and back of thighs. Fibrillation of calf muscles. Reflexes: Left ankle jerk absent, plantar responses flexor. Sensation normal.

General.—Venules over cheeks. Pyorrhœa. Heart, nothing definite. Blood-pressure 120/80. Urine: nothing abnormal. Spine: movements very poor; rigidity, mainly in the lumbar region; pain on bending to either side; tenderness over sacro-iliac synchondroses.

4.8.31.—Blood: Wassermann reaction negative. Cerebrospinal fluid: Wassermann reaction negative. Total protein 0·025%. Cells 3 per c.mm. Lange 010.

20.8.31.—Skiagram of spine: shadow between bodies of first and second lumbar vertebræ on their right sides. Examination of patient over this region reveals resistance and tenderness.

Hodgkin's Disease with Signs of Involvement of the Spinal Cord.—
—L. P. E. LAURENT, M.R.C.P.

J. E. M., married man aged 44, was admitted to the Hospital for Epilepsy and Paralysis, Maida Vale, on July 30, 1931, under the care of Dr. Russell Brain.

History.—Lassitude, anorexia and night sweats for six weeks. Frequency of micturition with some dribbling for three weeks.

Past history.—Nothing of importance except "bronchitis and pleurisy" two years ago.

Condition on admission.—A thin, pale-looking man. Nervous system: No abnormality detected in the cranial nerves and in the upper limbs. Lower limbs: Bilateral increase of tone and a little impairment of power at all joints, especially on the left side. Knee- and ankle-jerks increased equally on both sides. Plantar responses: Left extensor; right doubtful. Abdominal reflexes: None obtained. Sphincters: Within twenty-four hours of admission he developed complete retention of urine and has passed no urine without catheterization while in hospital. No sensory changes detected. Other systems: Numerous elastic discretely enlarged lymphatic glands in the left posterior triangle of the neck. Slight enlargement of inguinal lymphatic glands. Spleen: Not palpable. Chest: Harsh breath sounds, increase of vocal resonance and some râles at the left apex and left base.

Investigation.—11.8.31. Gland excised from neck. Pathologist reports "Hodgkin's lymphogranuloma."

Lumbar puncture was performed on 20.8.31, and again on 15.9.31. On each occasion a rapid rise of pressure took place on jugular compression. The cerebro-spinal fluid showed a rise of total proteins to 0·06%, but was otherwise normal in every respect, the Wassermann reaction being negative in the blood and cerebrospinal fluid.

Skiagram of spine.—No abnormality.

A four-hourly temperature chart has shown an occasional evening rise to 99° F. and once to 100° F.

A blood-count on 1.9.31 shows 3,420,000 red cells, 70% Hb. and 18,300 white cells, the differential count being within normal proportions.

Course.—The patient has received arsenic by mouth. His general condition has improved somewhat. His colour is better. Retention of urine still necessitates catheterization. The legs are rather weaker, and the plantar responses are both definitely extensor.

Disseminated Sclerosis with Marked Sensory Loss.—S. W. HARDWICK, M.D.

C. W., female, aged 19. Admitted to Maida Vale Hospital, 26.8.31.

History—Three years ago: weakness of left leg; away from work for nine weeks.

Two and a half years ago: diplopia for a few days.

One year ago: diplopia, nystagmus, intention tremor, loss of postural and vibration sense in legs. (In-patient at this hospital for six weeks.) Complete recovery.

Five months ago: sudden onset, stiffness left leg and weakness right arm. In-patient at Middlesex Hospital until 3.6.31, when she was discharged fully recovered.

Three weeks ago: noticed stiffness in right leg in the evenings. Two days later noticed left leg similarly affected.

On examination.—Euphoria. Pallor temporal halves of fundi. No nystagmus. Loss of power in right arm and much increase of tone. Co-ordination in left arm good. Loss of power and increase of tone, both legs. Reflexes: Tendon reflexes increased; abdominal reflexes absent; bilateral ankle clonus; plantar responses extensor. Slight analgesia over right hand and forearm, otherwise sensation normal. No loss of sphincter control.

31.8.31.—Complained of diplopia. Slight left ptosis. Anæsthesia and analgesia below the sixth dorsal segment on the left side. Diminished sense of position in the lower limbs, left > right. Incontinent.

14.9.31. Nystagmus. Right facial palsy. Slight right ptosis; weakness of right external rectus. Anæsthesia and analgesia over left side as above.

2.10.31.—The right facial palsy has now cleared. Gross inco-ordination and slight weakness of left upper extremity. Power still further reduced in lower limbs: no voluntary power in right limb, and only slight flexion of hip and great toe on left side. Complete anæsthesia and analgesia over the lower limbs and the trunk up to the level of the sixth dorsal segment; loss of vibration sense and sense of position together with gross impairment of thermal sense over the same area.

Cerebrospinal fluid: Wassermann reaction negative. Protein, 0·01%. Cells, 2. Lange, negative. Normal response to Queckenstedt's test.

Blood Wassermann reaction, negative.

Skiagram of spine, negative.

Muscular Dystrophy, Pseudo-hypertrophic Type occurring in two Sons of the same Mother but different Fathers.—CHARLES PINCKNEY, M.B., M.R.C.P.

C. B., aged 9. His mother has noticed during the last few years that he has difficulty in climbing stairs.

On examination.—Intelligent boy. Characteristic "climbing" method of getting up from supine position. Slight lordosis in erect position. Only slight "waddling" gait on walking. Slight tendency to slip through hands on attempting to lift him by axillæ. Cranial nerves: No abnormalities. Arms: weakness and wasting of extensors and flexors of forearm, extensors more than flexor, and of triceps and biceps, triceps more than biceps; weakness and wasting of both deltoids and of pectoralis

major, right and left; marked atrophy of latissimus dorsi; serratus magnus, supra- and infra-spinati, good power, no wasting; erector spinæ good power. Legs: pseudo-hypertrophy of calves with good power, circumference 9 in.; weakness of dorsi-flexors of feet; weakness of quadriceps with wasting; slight weakness of glutei.

No loss of sphincter control. No sensory changes. Reflexes: Tendon jerks absent in arms; abdominals present and equal; knee-jerks present and equal; ankle-jerks present and equal; plantars flexor.

E. S., aged 16. Shows a more advanced degree of generalized wasting and loss of power than his younger step-brother. In last ten years has noticed progressive weakness of legs; was able to take part in games at school until four or five years ago.

On examination.—Intelligent boy. Characteristic "climbing" method of getting up from supine position. Marked lordosis in erect position. Marked "waddling" gait on walking.

Cranial nerves.—No appreciable disease.

Arms.—Weak grip; weakness of extensors and flexors of forearm, with wasting, extensors more affected than flexors; wasting and weakness of triceps and biceps, triceps more affected than biceps; weakness of both deltoids and pectoralis major; atrophy of latissimus dorsi; serratus magnus, supra- and infra-spinati normal; weakness of erector spinæ.

Legs.—Pseudo-hypertrophy of calves; weakness of dorsi-flexors of feet; weakness and wasting of quadriceps and glutei. Measurement.—Calf, right and left, 11½ in.; thigh, right and left, 11½ in.

No loss of sphincter control. No sensory changes.

Reflexes.—Tendon-jerks absent in arms; abdominals present and equal; knee-jerks absent; ankle-jerks present; plantars flexor.

Syndrome of the Jugular Foramen.—C. WORSTER-DROUGHT, M.D., and T. R. HILL, M.D.

Mrs. A. T., aged 50, complains of huskiness of voice and occasional difficulty in swallowing (nine months' duration), also inability to lift left arm to usual height (six months' duration).

Previous history.—Right breast removed nine years ago. (Excision for ? cyst.)

Physical examination.—Pupils, fundi, and ocular muscles normal; speech hoarse with partial aphonia; left vocal cord in abducted position and paralysed; right vocal cord normal. Palate deviates to right. Atrophy and paresis of upper half of left trapezius and left sterno-mastoid. Tongue deviates to left on protrusion and left half is atrophied. Sensation normal. Knee- and ankle-jerks exaggerated. Other reflexes normal.

Cerebrospinal fluid, normal. Blood: Wassermann reaction, negative.

X-ray examination of skull in lateral view shows two small patches of calcification one above petrous bone and other below this. Basal view shows some translucency in neighbourhood of left jugular foramen.

Commentary.—The patient shows paralysis of the cranial nerves (tenth and eleventh) passing through the left jugular foramen and also of the left hypoglossal nerve. The operation scar and the fact that the axillary glands are intact does not suggest that the operation carried out on the right breast nine years ago was for carcinoma.[1] Consequently the presence of a secondary deposit in the jugular foramen need scarcely be considered. The nature of the localized lesion responsible for the paresis lies between "polyneuritis cranialis" and a neoplasm. The latter appears more likely, but as the growth appears to be stationary it is probably a tuberculoma.

1 It has since been confirmed that the right breast was removed for local cystic mastitis.

Congenital Bilateral Ophthalmoplegia and Facial Paralysis, with other Congenital Defects.—I. M. ALLEN, M.D.

A. C., male, aged 10 years and 4 months.

History.—Full-term child, confinement rapid, presentation vertex. At birth the fingers were joined and the feet turned in. During infancy the head was believed to be large. When four weeks old he seemed to become drowsy and did not look about. When seven weeks old his mother was told that he was blind. Until seven months old he made few sounds, simply lying and rolling his head from side to side. At seven months he improved in health and put on weight. When 12 months old an orthopædic operation was performed on his feet, which were kept in plaster for 15 months. Cut first tooth at 18 months, walked first at two years, and talked first at three years. Until then had made very few sounds, and seemed to have no feeling as he made no attempt to remove anything which must have irritated him. Until three years old he walked with his head down and made no attempt to look up.

At 5 years he had two attacks of convulsions, in both of which he twitched and foamed at the mouth. When 8 years old he had an operation on his webbed fingers and his tonsils and adenoids were removed.

Before he was eight years old he failed to learn his letters though attempts were made to teach him. He was not fond of pictures. Having been tried without success at an ordinary school, he was sent to a special school when eight years old. There he is learning very slowly. At first he wrote everything upside down, and later wrote letters from the right side of the page from right to left in a mirror fashion.

Two weeks ago he had a typical epileptic fit, for which condition he came under observation.

There was nothing unusual in the family history.

Condition on examination.—The head is larger than normal, and the forehead overhangs the face. No "cracked pot" sounds can be elicited. The forehead and face are smooth and show no movement or expression when he talks or is pleased. The palpebral apertures are small.

Upward deviation of the eyeballs is imperfect, but when the eyes are fixed on an object and the head rotated forward, the eyeballs assume a higher position than with spontaneous upward deviation. When told to look to the right, the right eyeball fails to move while the left eyeball moves in a very short distance. Similarly, when told to look to the left, the left eyeball fails to move and the right eyeball moves slightly. When told to look down, both eyeballs rotate freely downwards and inwards. It is impossible to get the patient to look directly at an object if it is placed below the level of the eyes and a little to one side. The eyeballs converge normally when looking at a near object. The pupils are dilated and equal; react briskly to light and consensual stimulation; but show no reaction on convergence. The optic discs are normal, and the retina shows scattered black pigment though within normal limits.

On attempting to close the eyelids, only partial closure is possible, the eyeballs rolling upwards and inwards. No movement of the face occurs when talking, and pleasure is expressed by a laugh without any movement of the facial muscles. Mouth small. Tongue small and moulded to shape of mouth. Speech is rather indistinct but readily understood. Other cranial nerves normal.

Motor and sensory functions normal; the tendon reflexes present and equal; and the plantar reflexes flexor. Other systems negative.

The child shows a moderate degree of obesity of general distribution. There are scars resulting from an operation to correct webbing of the first and second fingers on both hands. The feet are held in a position of talipes equinovarus.

The patient is left-handed. He recognizes individual letters with ease, but cannot recognize a word of three letters; though he can do so if allowed to spell it aloud letter by letter. He recognizes without hesitation letters printed normally, mirror fashion or upside down. He reads "M" as "W" and vice versa. He recognizes sounds and words without any difficulty. He is bright and cheerful, and interested in everything about him. His behaviour suggests an average degree of intelligence for everything for which reading is not essential.

Summary.—Congenital bilateral facial and abducent paralysis with other defects of ocular movements, probably due to absence or imperfect development of the corresponding nuclei. Syndactylism. Talipes equinovarus. Congenital alexia. Hydrocephalus. Epilepsy.

Section of Odontology.

President—Mr. E. B. Dowsett, D.S.O., L.R.C.P., M.R.C.S., L.D.S.E.

[*October* 26, 1931.]

PRESIDENT'S ADDRESS.

Operative Procedure for Cysts of the Jaws.

By E. B. Dowsett, D.S.O., L.R.C.P., M.R.C.S., L.D.S.Eng.

I propose to put before you this evening some data of a series of cysts of the jaws that it has been my lot to operate upon both in my private and my hospital practice during the last few years, and to offer a few comments upon the same.

The first is a series of so-called dental cysts. In the last four years I have operated upon, by the radical and open method, over seventy cases. As far as I can discover there is so little consensus of opinion as to the best technique in the treatment of such cysts, and so little actually recorded on the subject, that I would like therefore to give you, in detail, my own general procedure and experience.

There are two distinct and recognized methods of dealing with these cysts, which I will call the "closed" and the "open." By the "closed" method, after a suitable gum-flap has been made, the outer wall of the cyst opened and the whole of the contents and the lining membranes dissected out, then the cavity is again closed by sewing up the gum-flap. This is a method I adopt nowadays only when the cyst is no larger than the top of my little finger (i.e., about 12 millimetres in diameter). It is distinctly rare to find a cyst of this size to operate upon as they seldom give rise to any symptoms at this stage and so are frequently overlooked. There are some operators I know who on occasions adopt this method for the larger type of cysts, examples of which I shall show you on the screen shortly, but my experience of such procedure with these is that, in spite of all possible precautions, the blood-clot necessarily left inside—because the cyst cannot collapse—frequently breaks down, with the result that there is a large suppurating cavity which either bursts through the stitched flap or has to be opened. This necessitates tedious syringing through but a small opening, probably for months, before this suppuration ceases. Therefore, my own procedure with all these larger cysts is to adopt the "open" method. To assist my description I show some rough diagrams (figs. 1 to 4). I have omitted teeth from these diagrams in order to simplify their appearance.

By this method I make a large oval or rectangular flap so as to be quite free of the lateral limits of the cyst cavity, and having the alveolar edge of the flap well to the outer side of the alveolar ridge so as to leave the ridge itself intact, and thereby not to interfere with the early insertion of a denture. This flap is then carried right down as far as possible into the sulcus by raising with a periosteal elevator if there is bone over the outer side of the cyst—or by careful dissection with a knife to free the gum-flap if the outer plate of bone has been absorbed in any spot—in order to leave the lining membrane of the cyst intact, if possible, and not bring part of it away on the gum-flap. The dissection is then carried on beyond the

depth of the sulcus, so as to free all the soft tissues from the limits of the whole of the outer side of the cyst. This often necessitates freeing the soft tissues right down to the lower border of the mandible or very high up in the maxilla. The cyst is then opened with a knife, burr or chisel, according to the consistency of the cyst-wall, at that part nearest the alveolar border, and as much of the cyst-wall—bone and lining membrane—removed as is already laid bare. This is most easily done with short-bladed crown scissors if the bone is thin or absent, but may necessitate

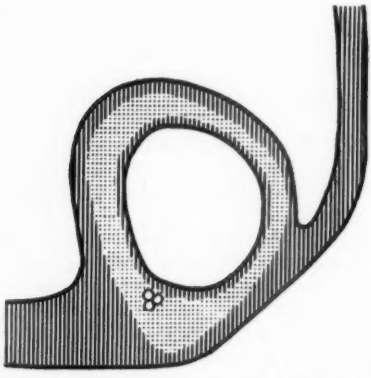


FIG. 1.—Transverse section of mandible in molar region showing large dental cyst.

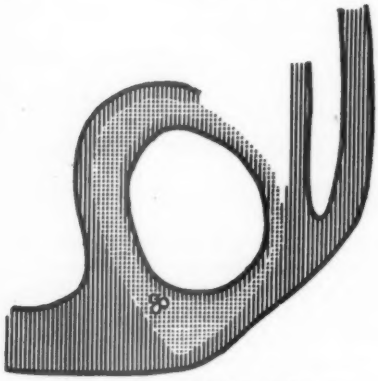


FIG. 2.—Gum flap dissected up.

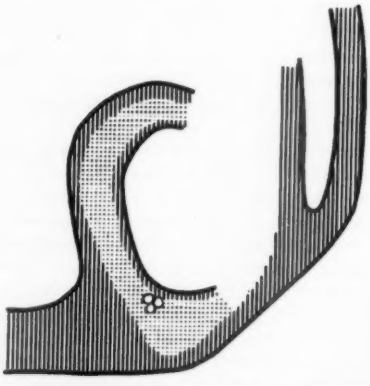


FIG. 3.—Dissection of soft tissues carried down to base of cyst and whole of outer wall removed.

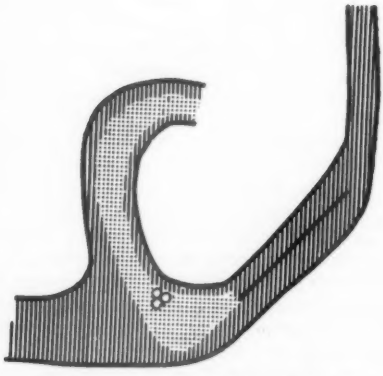


FIG. 4.—Gum flap tucked in and meeting lower edge of retained lining membrane of cyst.

a burr or chisel if the bone is thick. The contents can then be evacuated. The farthest part of the outer wall, which is beyond the level of the sulcus, is generally best removed "piecemeal" by means of bone-nippers—the whole object being to make the opening into the cyst as large as the greatest diameter of the cyst cavity itself.

Now there is another matter to be considered at this stage and about which opinions vary, and that is, as to whether the remainder of the lining membrane

should be removed. For reasons which I will explain later and which I think are very beneficial, I always leave the lining membrane intact on the inner wall of the cyst, unless I find that it is badly diseased with very long-standing suppuration. As the furthest limit of the outer wall has often to be removed with bone nippers, it is very easy inadvertently to tear away some of the lining membrane of the inner wall, where one wishes to leave it intact, and so to avoid this, where possible and expedient, I make a definite incision with a knife right through the lining membrane along the lowest margin of the cyst cavity in the mandible and the highest margin in the maxilla. This quite prevents any tearing up of the lining membrane and makes it easy to remove that portion on the outer side of the incision. The gum-flap, which all this time has been held away either by means of a long ligature passed through it or by a retractor, is now turned right inwards, so that the raw surface of the flap lies against the raw surface dissected away from the cyst wall beyond the limit of the sulcus. My object, theoretically, is to make the edge of the flap exactly meet the cut edge of the lining membrane and this may necessitate slight trimming, but more generally there is a slight deficiency rather than an overlap. In this way the whole bony cavity, which is now saucer-shaped, is open to the mouth, and either completely lined or very nearly lined with epithelium. Any slight deficiency in this respect very soon becomes epithelialized over and thus any further exudation is prevented. If on the other hand there should be a slight overlap of the flap by its being too large, it is of no consequence, as it very soon contracts off the epithelial lining and only gives rise to a slight pucker. After tucking in the gum flap I always pack the cavity lightly with sterile gauze for twenty-four hours in order to keep the flap in position, after which no further packing is required. This packing is generally removed with very little discomfort by first soaking with weak hydrogen peroxide. It is very seldom that any excessive hæmorrhage occurs in operating upon these cysts, and what there is, is easily controlled with a little pressure or with hot saline.

It is often said that when the whole of the lining membrane is removed and the cavity left bare into the mouth, such cavity granulates up from the bottom and thus becomes obliterated. My decided opinion is that nothing of the sort ever happens. Now how does this obliteration take place? My own observations show that it seems to be definitely due to the growth of bone from the whole of the remaining surface of the cyst cavity, *under the epithelium*. By the operative procedure I have described, any small raw surface or denuded bone at the alveolar edge or at the base of the cyst, very rapidly gets epithelialized over and it is then that the bone soon begins to be deposited. This bone can easily be demonstrated, both by actual measuring and also radiographically. It is in consequence of this that I strongly advocate leaving the epithelial lining upon the inner wall and also the tucking-in of the gum-flap, so that in the minimum of time the whole cavity left opened up is covered with epithelium. If the lining membrane on the inner wall is removed, then the whole surface has first of all to become covered with epithelium by growing in from the edges, before any growth of bone and obliteration of the cavity can take place. Therefore, theoretically there should be a considerable delay in the filling-up process when this latter method is adopted. Practically in my experience this is exactly what happens; and the bone does begin to grow out more quickly if the epithelial lining is left intact on the inner wall of the cyst and considerable time is saved in the general obliteration process.

Amongt the various workers and writers on the subject of regeneration of bone, such as Keith, Adami, Macewen, Ham, Greig, Phemister, Leriche, Policard and many others, there is no definite opinion as to how such bone is re-formed. Of the various cells in association with the bone it is generally conceded that the adult bone-cell takes no part in regeneration. Therefore the remaining bone-cells to consider are the osteogenic cells or osteoblasts found in the Haversian canals and

in the periosteum. Those found in the endosteum do not concern us here in the jaws. It is just possible that the osteogenic cells in the Haversian canals might take some part in re-forming bone on the inner side of the cyst, but it is more likely that there are no Haversian systems in that situation. The osteogenic cells in the periosteum can only be at the edge of the remaining cyst cavity, and therefore would have to grow centripetally under the remaining lining membrane before they could form bone, and this would take a long time. Therefore it seems likely that the regeneration of bone at the base of the cyst takes place by the process of so-called "metaplasia." Adami defined metaplasia as follows: "Metaplasia is the post-natal production of specialized tissue from cells which normally produce tissues of other orders and is an adaptation on the part of the cells to altered environment." There are two kinds of cells that will be found under the remaining lining membrane that, according to this theory, might take on the function of bone formation, namely, the fibroblasts and the osteoclasts—the latter having made their appearance in order to absorb the bone away during the cyst formation—and by a metamorphosis or by a change in function, due to the altered environment, either of these may deposit bone. As a sidelight upon this we continually see rarefaction and deposition alternating in pathological conditions of both bone and cementum, either for no apparent reason, or because the character of the pathological condition changes locally—in other words there is an altered environment.

Another great advantage in leaving the lining membrane on the inner wall and tucking in the gum-flap is that there is only a very short period, at most a few days, when there is any discharge from the cavity, because the very small granulating surface left at the alveolar edge, or at the junction of the tucked-in flap with the remaining lining membrane, is so very rapidly epithelialized over that there is no surface left to exude. The practical matter of syringing is thus reduced to a minimum and the cavity is very easily kept clean by the patient himself. Also, because of the absence of discharge, and because the alveolar ridge has been left intact, dentures as necessary can be inserted after a week or two at most. On the other hand, if the whole lining membrane is removed and no flap tucked in, there is a serous or purulent discharge from the raw surface for a long period, often extending over many weeks, or even months, which needs constant attention.

Still another advantage in removing the *whole* of the outer wall of the cyst and tucking in the gum-flap is that it almost entirely prevents the contraction of the opening made into the cyst. If the outer wall of the cyst is removed only as far as the level of the sulcus and therefore no position made for the gum-flap to be tucked in, there is a great tendency for the orifice to contract much in advance of the obliteration of the cyst cavity, and so there is a corresponding tendency to hold up food and also a great difficulty in cleansing.

With regard to the healing process in these cysts in the maxilla, it is often said that there is no regeneration of bone in that situation after it has been removed. From my observations it is most definitely re-formed, though at a slower rate than in the mandible.

Many of the larger dental cysts in the premolar and molar regions in the maxilla invade the region of the antrum so that the actual membranous cavity is often much reduced in size and in some cases completely obliterated by the outer wall of the antrum being pushed right up to and in contact with its inner wall. I have operated on many cases of this latter nature. In one particular case, during the operation, when the whole of the outer wall of the cyst had been removed and I was trimming up the edges, a very striking phenomenon occurred. The lining membrane on the inner wall of the cyst had been left intact as usual, when, in consequence apparently of an increase of pressure in the nasal cavity from the anæsthetic, and because all the bone on the outer side of the antrum had been

absorbed, the antrum suddenly blew out like a small air balloon and completely filled the cyst cavity, and then by the varying nasal pressure this was sucked in and blown out again several times. The after-history of this case was most interesting and instructive, and perhaps remarkable. I packed the cyst cavity very lightly for twenty-four hours, just sufficiently to keep my usual gum-flap in position, and healing was rapid and uneventful. But the interesting point is that bone actually grew again between the lining on the inner cyst wall and the lining membrane of the antrum and in a few months the condition was apparently quite normal.

This condition of affairs, therefore, where the lining membrane of the cyst may be in actual contact with the lining membrane of the antrum, but back to back so to speak, with no bone intervening, is another very good reason for leaving the epithelial lining on the inner wall of the cyst, and its removal would almost certainly open the antrum and complicate the condition considerably and quite unnecessarily.

I will now show you some skiagrams of a few representative cases of these cysts, and some showing regeneration of bone after operation (slides 1 to 30).

An interesting point I have noticed in many of these cysts is that when large they are often not quite globular in outline, as one would expect, but appear to be partly loculated. Now this may be due to a peculiar burrowing propensity on the part of the growing epithelium (slides 2, 3, p. 7), but it also seems that in some cases (slides 4, p. 7, and 6, p. 8) it may be due to the original cyst, during its growth, encroaching upon the root of another tooth and thereby starting the epithelial remnants in the periodontal membrane of that tooth to grow and form another cyst which afterwards coalesces with the original cyst. This apparently may occur even when the pulp of that tooth is alive, as in the cases shown. This point is confirmed also by two cases I brought before this Section several years ago of dental cysts around the roots of living teeth, which seemed to have arisen around those teeth and were not merely encroaching cysts. This is not at all in conformity with the work and deductions of Warwick James and Counsell, who have shown that the epithelium in granulomata and cysts arises from the gum epithelium which has grown in via a sinus from a "dead" tooth; but at the same time these observations do seem to point to the possibility of the epithelial remnants in the periodontal membrane taking on active growth, though I can show no histological evidence to that effect.

I will now describe to you a few unusual forms of dentigerous cysts that I have had to deal with.

The first one (slide 31, p. 9) is that of a dentigerous cyst of a maxillary supernumerary incisor. In teaching the diagnosis of a dentigerous cyst one had always mentioned that a tooth would generally be missing from the arch, but not necessarily, as it might be growing from a supernumerary, but until I came across this one in 1926 I had never observed one nor seen one described. Since that date, however, I have seen three further cases described. This one, as its appearance indicates, was simple to deal with and healed rapidly.

The second case (slide 32) is a dentigerous cyst of a right mandibular third molar. Incidentally such cysts in this situation are fairly common, and I have operated upon six in the last five years. The interesting point about this particular case is the fact that the whole of the crown was not free into the cyst as has always been the case with dentigerous cysts in my experience. As is seen from the skiagram only about one quarter of the crown is exposed to the cyst cavity, the remainder of the crown being deep in and surrounded by bones and gum tissue. Also the tooth is partially inverted and the under surface of the root is denuded of bone, but in contact with the lining membrane of the cyst. It might be argued that this was a dental cyst caused by infection from the surface behind the second molar, but it was so deep as to preclude this suggestion, in my opinion. This case I operated upon

by the usual method after removing the tooth. It healed readily, and the regeneration of bone was rapid (slide 33).

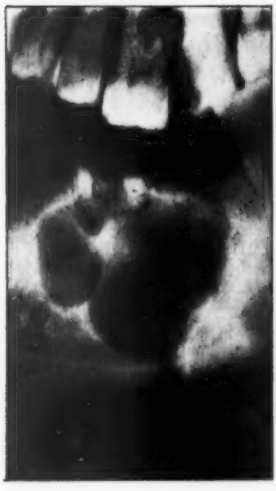
The third case (slides 34, 35 (p. 7), 36) is what one might call a compound dentigerous cyst, if I may suggest a new term in the nomenclature of such cysts. It had the crowns of three teeth, all presenting into and uncovered in the same cyst, the roots being fixed in varying degree in the bony wall. As far as my experience goes this is quite unique. There was also one other unerupted tooth buried in the bone immediately adjacent to the cyst. The cyst was in the middle line of the mandible, and the three teeth presenting into the cyst were the right second incisor, a right supernumerary incisor and a left supernumerary incisor, the buried tooth being the left second incisor. The lowest tooth of all, namely the right second incisor, had its apex presenting through the lower border of the mandible and not covered by any bone. This is well shown in the skiagram, and could be easily felt with the finger. It might be argued that this case started as a dentigerous cyst upon one tooth, possibly the lowest one, and that the other teeth presenting into it were merely unerupted teeth laid bare by absorption of bone during the enlargement of the cyst, but from the extreme similarity in which all three crowns were presenting into the cyst, one is rather forced to the conclusion that it was a case of simultaneous development of dentigerous cyst upon all three teeth, and that these three original cysts eventually joined up into one. There are are no special points to recount in the operation. In order to assist I removed the two first incisors, and the healing and obliteration were normal. These teeth were low down and the cyst so large that I thought it inadvisable after opening the cyst to leave either of the teeth involved in position, as advised by Sprawson in some cases. As the patient was of middle age, it seemed unlikely that the teeth would ever rise into position.

With regard to the histological examination of the lining membranes of these cysts, both dental and dentigerous, all those cases that I have examined have had the quite normal appearance of two or three layers of small epithelial cells with occasional bare patches.

I may add finally that every one of these cysts upon which I have operated has become completely obliterated, and in a space of time varying from three months to two years each one has resulted in either a mere depression on the outer wall of the jaw in the sulcus or has become so completely obliterated as not to be recognizable at all other than by a slight puckering of the mucous membrane.

Slides Shown.

1. Small dental cyst of mandible. Suitable for operation by "closed" method.
2. Large dental cyst of mandible—somewhat loculated.
3. Dental cyst of mandible—loculated.
4. Large dental cyst of mandible, originating from first molar which has been lost, but with special extensions under the second premolar and the second molar, both of which teeth had living pulps.
5. Showing regeneration of bone after six months in case shown in slide 4.
6. Large dental cyst of mandible originating from second molar root, with special extension under second premolar, which had a living pulp.
7. Showing regeneration of bone after eight months in case shown in slide 6.
8. Dental cyst of mandible.
9. Showing regeneration of bone after seven *weeks* in case shown in slide 8.
10. Dental cyst of mandible.
11. Multiple dental cysts of mandible.
12. Multiple dental cysts of mandible.
13. Dental cyst of maxilla in incisor to canine region.



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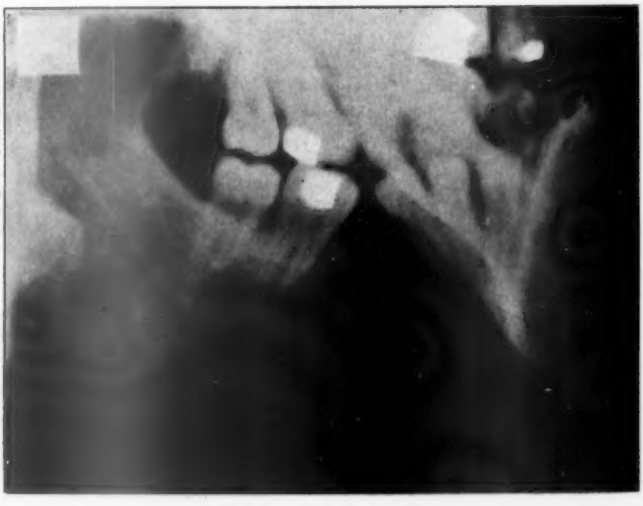

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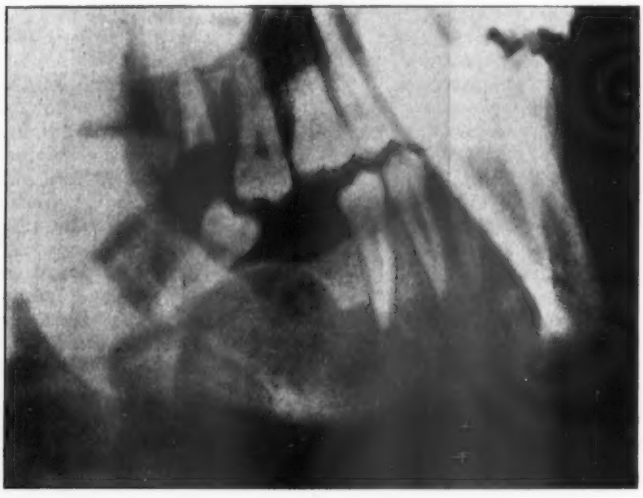

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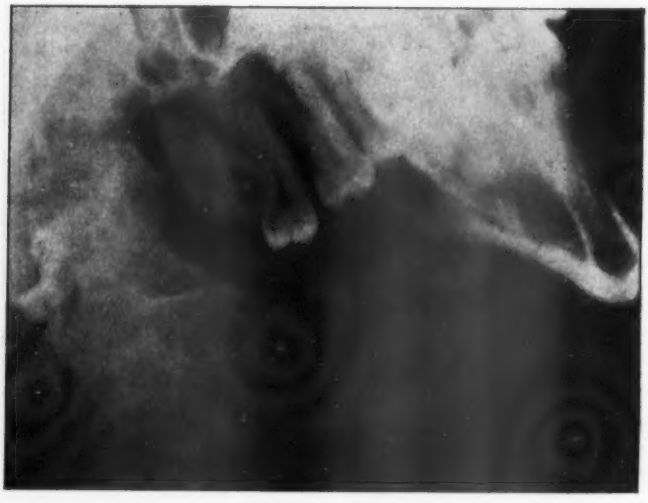

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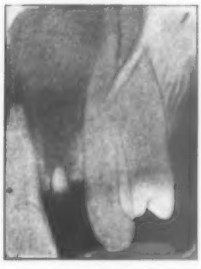
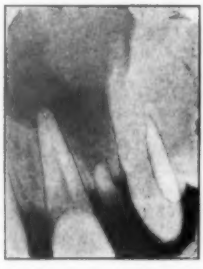
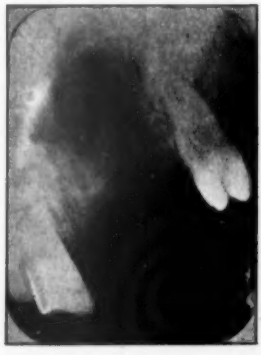
14. Showing regeneration of bone after three months in case shown in slide 13.
15. Dental cyst of maxilla in incisor to canine region.
16. Showing regeneration of bone after ten months in case shown in slide 15.
17. Dental cyst of maxilla from right incisor to left canine.
18. Showing regeneration of bone after ten months in case shown in slide 17.
19. Dental cyst of maxilla from right incisor to left canine.
20. Dental cyst of maxilla in premolar region just encroaching upon the antrum.
21 and 22. Large dental cyst of maxilla from left central incisor to first molar, almost obliterating the antrum and burrowing across the palate.







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23. Showing regeneration of bone after fifteen months in case shown in slides 21 and 22.
24. Dental cyst of maxilla in premolar region, almost obliterating the antrum.
25. Showing regeneration of bone and re-formation of the antrum in case shown in slide 24.
26. Large dental cyst of maxilla from incisor to premolar.
27. Showing regeneration of bone after four months in case shown in slide 26.
28. Dental cyst of maxilla in first molar region, completely obliterating the antrum.

29. Dental cyst of maxilla in first molar region, completely obliterating the antrum.
30. Dental cyst of maxilla in second molar region encroaching upon the antrum.
31. Dentigerous cyst on supernumerary maxillary incisor.
32. Dentigerous cyst on right mandibular third molar.
33. Showing regeneration of bone after four months in case shown in slide 32.
34, 35, 36. "Compound" dentigerous cyst of mandible in incisor region.

Section of Ophthalmology.

President—Mr. ELMORE W. BREWERTON, F.R.C.S.

[*October* 9, 1931.]

Pseudo-Glioma of the Retina. Massive Exudate.—ADRIAN CADDY, F.R.C.S.

This patient, C. B., a boy, aged 8, has not complained of any symptoms, but his mother noticed a white reflex from the pupil of the left eye in May, 1931. He had measles two years ago; otherwise his health has been excellent. The Wassermann reaction is negative.

On examination.—A large white exudate is seen in the vitreous, arising from the outer side of the disc, and gradually extending to the nasal inferior quadrant of the retina with the formation of fibrous bands detaching the retina.

No treatment has been adopted. The question may arise as to whether the eye should be enucleated if the mass becomes larger.

Megalophthalmos.—FRANK W. LAW, F.R.C.S.

The patient, aged 23, came to me complaining of defective right vision; he stated that this had always been so. The right globe is seen to be enormous; it has about 25D of myopia and 5D of myopic astigmatism, axis down and out. There is a good anterior chamber; many vertical ruptures of Descemet's membrane are seen. There is a scar on the right temple which the patient says was caused by a birth injury; is there possibly a causal connection between this and the eye condition?

Thomson and Buchanan [1] have described many cases of ruptures of Descemet's membrane through birth injury, but none with such high myopia. More lately, Vail [2] has described a condition of "megalophthalmos sine glaucoma," which he differentiates from buphthalmos. The list of possible causes enumerated by him does not help us here, though it may be significant that 44 out of his 92 cases were myopic.

References.—[1] *Trans. Ophth. Soc.*, 1903, xxiii, 296. [2] *Arch. of Ophth.*, July, 1931, vi, 39.

Coats' Disease.—FRANK W. LAW, F.R.C.S.

E. T., aged 12, came to University College Hospital in July, 1931, for refraction. The eyes were externally normal, but left vision was found to be "fingers at 1 metre," with poor fixation. There is a mass of white exudate at the macula, connected to the optic disc by fine strands (this may, however, be simply an optical effect). There is some pigment on the upper part of the mass, and there are some small deposits in the peri-macular region, and many scintillating deposits in the upper periphery.

In August the peri-macular deposits were found to have increased in number and size, and again in September. An attempt to take blood for a Wassermann reaction failed.

I think that this is rightly labelled "Coats' Disease": it compares very well with Coats' third case in his series quoted in the original paper.

I am indebted to Mr. Humphrey Neame for his consent to my showing this case.

Aberrant Cilia.—F. W. LAW, F.R.C.S.

D. D., a boy, aged 7 years, first seen in May, 1931, came to me for refraction; his mother said he had always had an extra bunch of eyelashes; apparently he was born with them. I have never seen anything like the condition before, and I think it is a dermoid. The hairs exactly resemble the cilia of the lid margin; there is a bunch of six or eight springing from a pocket in the apparently normal skin about half-way up the upper lid.

Episcleritis due to Focal Sepsis.—A. F. MACCALLAN, C.B.E., F.R.C.S.

I showed this case in February last (for previous notes see *Proceedings*, 1931, xxiv, 889, Sect. Ophthal. 33). As only moderate improvement followed the operation on the left antrum, it was repeated, and bilateral turbinectomy was also performed. Within a few days the patient's condition began to improve, and has continued to do so ever since. There is now no pain or discomfort, and vision is $\frac{6}{6}$ in each eye. There has been no ocular treatment, except a few drops of normal saline solution. The condition was an episcleritis, which has been cured by the elimination of focal sepsis.

Some Selected Cases of Reconstructive Surgery in the Orbital Region.—T. POMFRET KILNER, F.R.C.S.

I propose to demonstrate fifteen cases of plastic work about the orbit by means of photographic records.

The first seven of these cases have been exhibited here to-day and will be dealt with in some detail.

Case I (fig. 1) illustrates the use of the Thiersch graft for the reconstruction of an eye socket sufficiently large to allow of the wearing of an artificial eye of normal size.

This patient gave a history of injury to the right eye at eight years of age. The eye was removed when she was twelve years old. No artificial eye was worn until she was twenty years old when attempts to fit one proved unsatisfactory and a skin-grafting operation in the lower sulcus region was carried out in Nottingham.

When I first saw her she was aged 31, and was wearing a very small artificial eye which looked almost directly upwards. There was practically no sulcus above and very little below, except in the middle third where the remains of the previous skin-graft could be seen. The upper eyelashes were drawn in and lay on the surface of the eye. Through the lower eyelid there was a fistulous opening from which tears escaped.

The first pair of photographs show her appearance at that time. It will be noted that the eyelids cannot be approximated in spite of the very small size of the artificial eye.

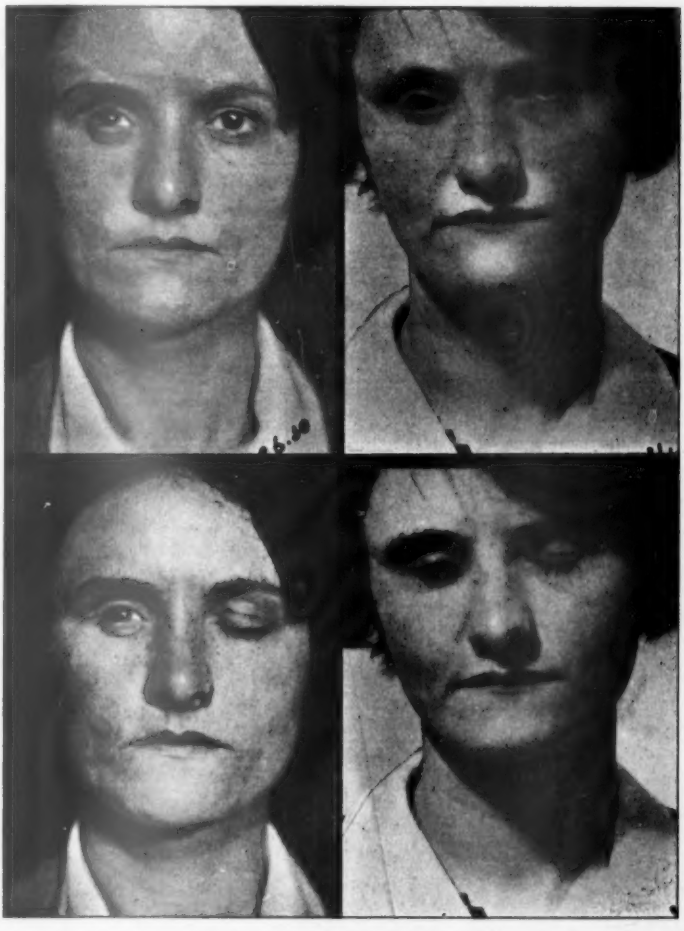
The second pair of photographs indicate her condition after freeing of the lids and reconstruction of the socket by means of a Thiersch graft applied on a large glass mould. The fistulous opening in the lower lid has been closed.

The artificial eye now worn is of normal size and the eyelids are readily approximated over it. The eyelashes no longer lie on the eye.

Case II.—(Photographs of this case were published in "Recent Advances in Surgery," London, 1928).—The patient was referred to me from the Radium Institute by Mr. Hayward Pinch in 1924. She was then aged 38 and had suffered from lupus of the face since she was eleven years old. Her early treatment had been by "scraping" and later she had had many years' treatment by radium.

There was much scarring of the left cheek and many telangiectases were present. The eyelid was dragged down and out. In one region active ulceration was still present though it was difficult to say whether this was true lupus or the result of treatment.

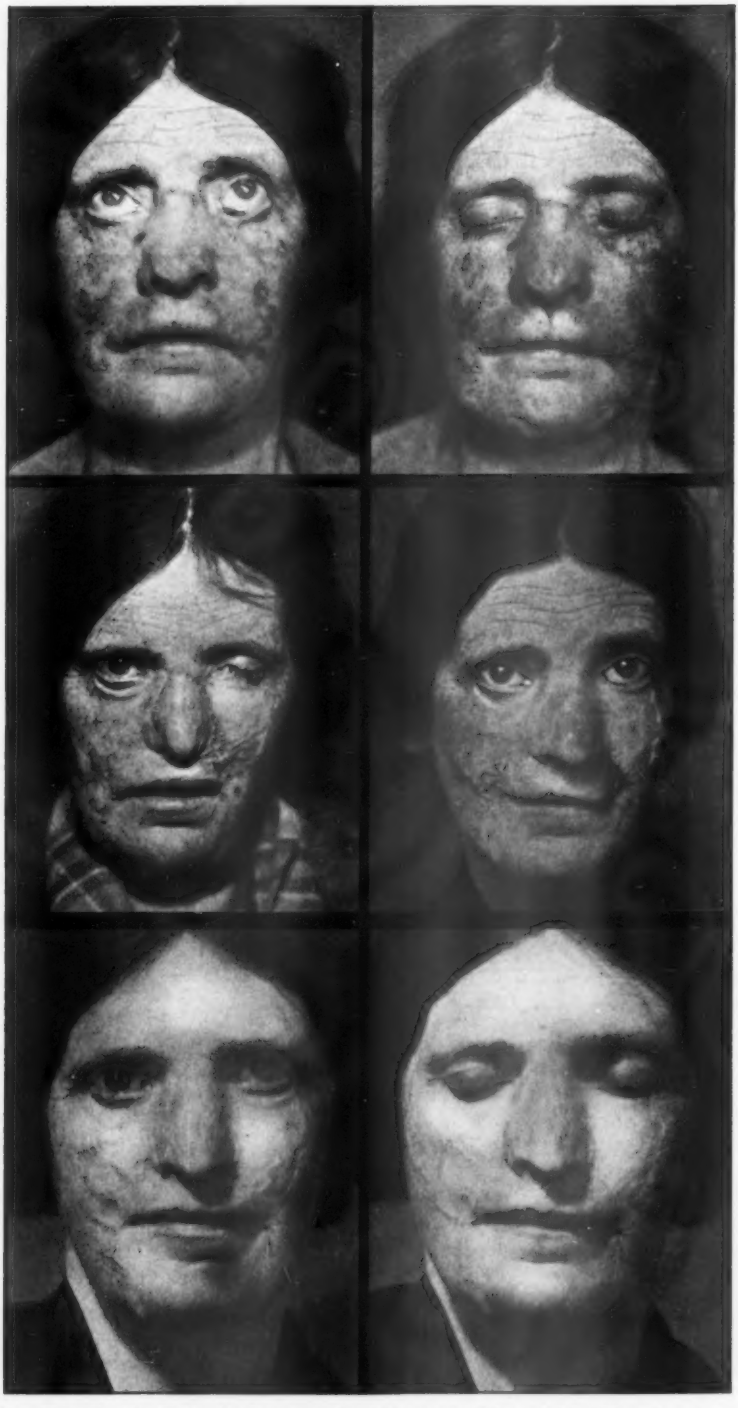
In this case I followed what I consider the soundest principle in such cases, when the outlook is doubtful—namely, the replacement of the diseased area by the most surgically economical means at one's disposal, the Thiersch graft. I freed the lower eyelid, excised the scarred area of the left cheek and applied a single large Thiersch graft on a mould.



Case I. FIG. 1.—Obliteration of upper fornix preventing approximation of eyelids even over an ill-fitting eye of very small size. Reconstruction of eye-socket by Thiersch grafting.

The graft took well, restored good function to the eyelid and, as you have seen, gave also a most gratifying cosmetic result.

In this case there occurred further ulceration, but not in the grafted area. Dermatologists are inclined to oppose the excision and grafting of old lupus areas because they fear the stirring up of local disease. In this case the only further



Case III. FIG. 2.—Extensive lupus scarring of face treated by multiple Thiersch grafts to eyelids, nose, upper lip and cheeks.

ulceration was anterior to the graft and this area reacted favourably to excision and grafting. Recently I have excised a scarred area from the neck and you will have noted that this area is still rather wrinkled. Grease massage is being carried out here and the cosmetic result in this region is likely to be every bit as good as that obtained in the face.

Case III (fig. 2).—This patient had suffered from extensive lupus of the face since she was 11 years old. At the time of my first examination, when she was referred to me from the Radium Institute by Mr. Roy Ward, she was thirty-four.

She had undergone a great variety of treatment and, during the last year, had been having radium treatment at two-monthly intervals. This had produced improvement, but there were still several areas of ulceration and much brawny swelling of the upper lip. It was difficult, again, to say whether these were true lupus or, in part at any rate, the results of treatment. There was ectropion of both lower eyelids, more marked on the left than the right side, and epiphora was very troublesome. The latter had persisted in spite of incision of the canaliculi.

I commenced treatment in this case again by the surgically economical replacement of scarred areas by Thiersch grafts. The ectropion on the right side was treated on the usual lines: incision from canthus to canthus, just outside the eyelashes, gradual division of all scar tissue bands, and the application of the graft on a small Stent mould held in position by sutures which everted the lid over the mould and so pressed the graft home on the raw surface. On the left side I freshened the lid margins in their middle two-fourths and sewed them together before incising and freeing the eyelids. On this side the graft was applied on a flat mould, for part of the cheek skin was excised at the same time.

The second pair of photographs indicates the appearance in an intermediate stage and illustrate the appearance of a recently grafted eyelid.

The third pair of photographs illustrates her appearance about fourteen days ago before my last intervention. The nose skin has been replaced by Thiersch grafts, as have also large areas of both cheeks and upper lip. Ten days ago I inserted a small fat graft under the Thiersch graft of the left eyelid to fill up the hollow in this region and a similar graft has improved the appearance of the bridge of the nose. Various other minor trimming plastics were carried out at the same time.

The points considered worthy of note in this case are: (1) The satisfactory functional result in the eyelid regions. (2) The great improvement in the patient's mental attitude towards life. (3) The complete absence of any suggestion of recurrence of lupus in the grafted areas.

Case IV (fig. 3, p. 6).—This case has been shown to illustrate the cure of severe ectropion by Thiersch grafting and the reconstruction of eyebrows by free full-thickness (Wolfe) hair-bearing grafts from the post-mastoid region. The case itself and its photographic records indicate well what an important part reconstruction of eyebrows plays in the cosmetic results in burn cases.

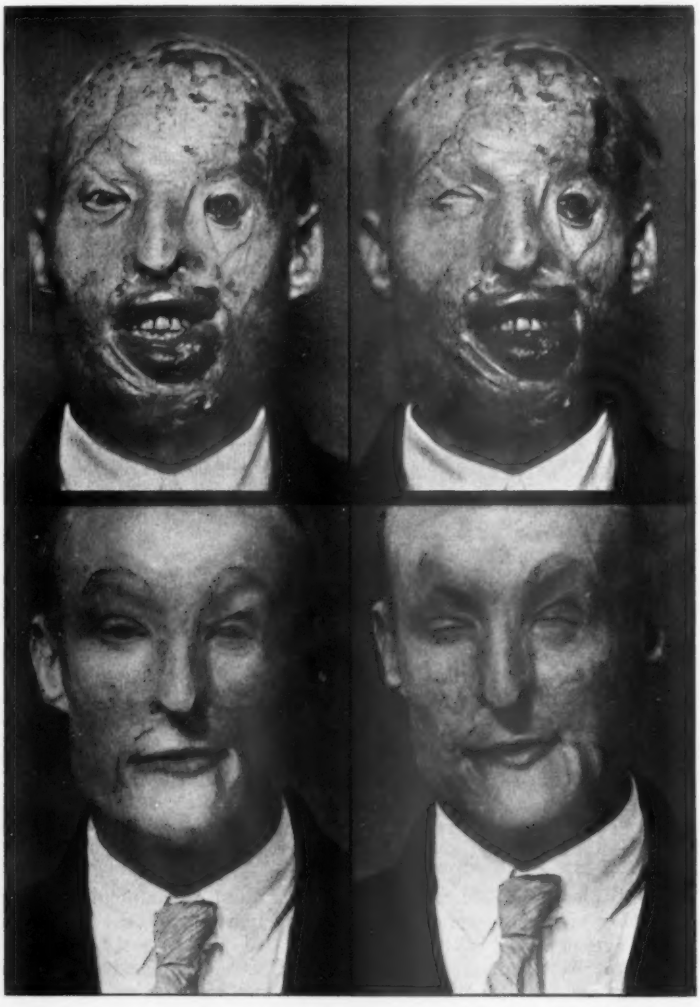
The patient was removing a bung from a drum of nitric acid. The bung was defective and flew off, the patient receiving a large quantity of acid full in his face.

The early eyelid grafts in this case were carried out as a prophylactic measure, for the left eye was dangerously exposed. Contraction occurred in these and further grafting was carried out at a later date. The eversion of the upper lip was treated in a manner precisely similar to that used for the eyelids.

The case also illustrates extensive reconstruction of the chin and lower lip regions by means of a Gillies tubed-pedicle flap brought up from the abdominal wall via the wrist in three stages.

Case V.—This was a case in which I felt justified in attempting the reconstruction of eyelashes by the insertion of a hairy Wolfe graft in the margin of the upper eyelid.

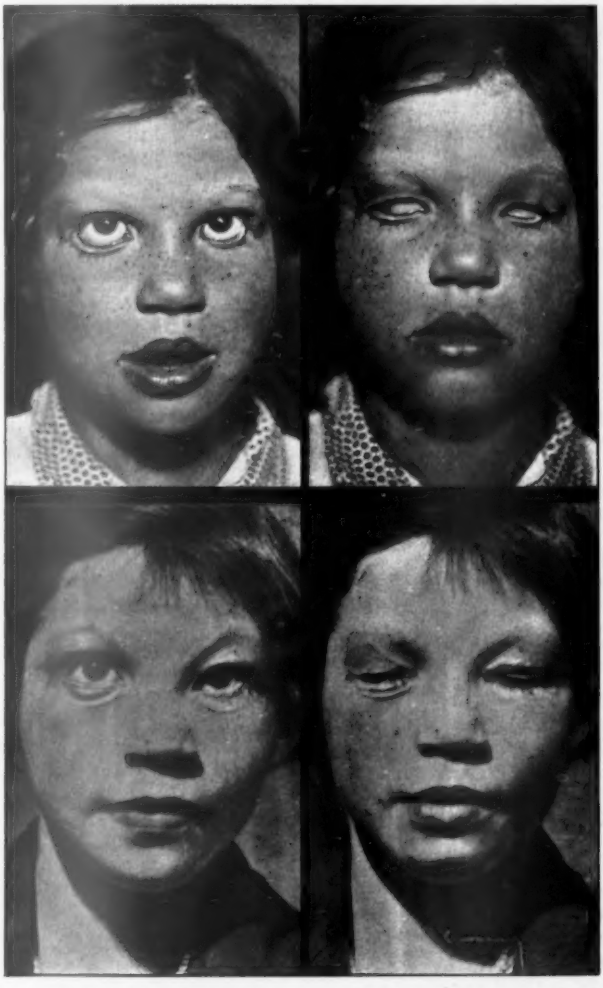
The injury was a burn of both right eyelids, sustained when the patient came in contact with a hot iron during an epileptic seizure. She was referred to me by Dr. Maccormac at St. James's Hospital, Wandsworth. The burnt areas were then



Case IV. FIG. 3.—Extensive nitric acid burn of face. Ectropion of eyelids and eversion of upper lip treated by Thiersch grafting: eyebrows reconstructed by hair-bearing Wolfe grafts: chin and lower lip reconstructed by tubed-pedicle flap from abdominal wall.

covered by healthy granulations, but contraction was already beginning. There was some haziness of the lower half of the cornea. As a prophylactic measure the margins of the eyelid remains were sewn together, the granulations gently

shaved off and a Thiersch graft applied on a mould. After separation of the lids, further contraction occurred and the lower half of the globe was left exposed. A new lower eyelid was reconstructed by means of a forehead flap, and at the same time a narrow strip of eyebrow skin was sutured in position in the split margin of the upper lid. Since that time I have attempted to reduce the thickness and



Case VII. FIG. 4.—"Congenital" shortage of eyelid skin treated by Thiersch grafting.

immobility of the lower eyelid, but the case still illustrates the disappointing results to be expected in reconstruction by full thickness *flaps* in this region.

The photographic records do not show the eyelashes, but a complete line of them is present. They grow freely and require frequent trimming. One cannot help feeling that, in a patient of different type, much more might be made of them with the aid of a suitable cosmetic.

Case VI.—This is primarily a jaw case in which I have carried out a bone graft from the iliac crest for the reconstruction of the right side of the mandible. The loss of bone was due partly to what was diagnosed as a sarcoma in childhood and partly to prolonged X-ray treatment which had destroyed all hair on the right side of the face. The scarred area of the right cheek has been replaced by a tubed-pedicle flap from the chest and the eyebrow has been reconstructed by a full-thickness hairy graft from the post-mastoid region. Comparison between the photographic records of the early condition and the patient's present condition indicates again the importance of eyebrow reconstruction in disfigured cases of this type.

Case VII (fig. 4, p. 7).—This case was referred to me in September, 1929, by Mr. Richard Colley, of Bath, with a history that shortly after birth there appeared eversion of the eyelids and some discharge from the eyes. When two years old the patient spent two months in hospital where, apparently, bilateral external canthorrhaphy was carried out. When she was five years old a further operation was performed on the left lower lid and she has been in hospital for minor operations on some four further occasions.

When I first saw her she was nearly eight years old and although the history left little doubt that the condition had improved, the lower eyelids were still very far down, everted, and sagging away from the globe. There were scars in both external canthus regions and one vertical scar in the centre of the right lower eyelid where a wedge-excision had evidently been carried out. The sight was stated to be good in both eyes. Muscle action appeared to be good and forcible closure of the eyelids expressed tears. There was obvious shortage of skin in a vertical direction in all four eyelids and normal closure of the eyes left the lower half of the globe uncovered.

Treatment of this case was carried out on the lines used for the cure of cicatricial ectropion—the eyelid margins being sutured together before applying the grafts on moulds. The right eye was treated first and the left after a period of two months, when it was considered safe to separate the marginal adhesion on the right side. Some contraction of the grafts occurred and further grafts were added to the right lower and the left upper eyelids. The photographic records and the case itself indicate the improved covering of the globe which has been obtained.

Case VIII.—A severe sulphuric acid burn of the face. Marked ectropion of the right upper lid. Remains of socket mucous membrane on the left side from which the eye had been removed.

The ectropion was treated by Thiersch grafting; the burnt skin of the nose was replaced by a non-hairy Wolfe graft, the left alar deformity being corrected at the same time; the deformity of the angle of the mouth and the eversion of the lower lip were corrected by a small tubed-pedicle flap from the neck.

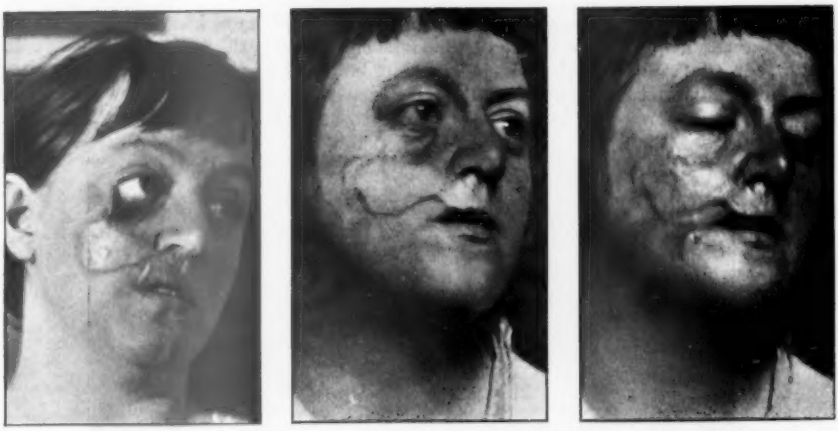
Case IX (fig. 5).—A case of severe downward drag on the right lower eyelid following a burn produced by falling forward on the fire in a faint. The patient was not an epileptic.

The photographic records show the cure of the ectropion by Thiersch grafting and also the correction of eversion of the upper lip by similar means.

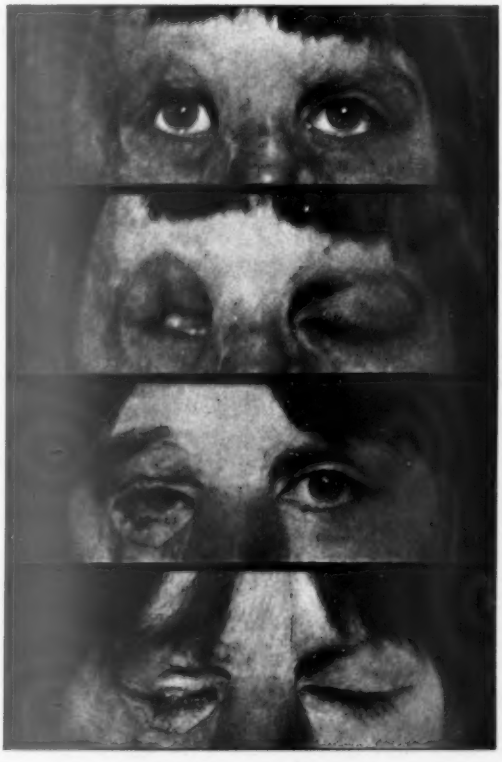
Case X.—A case of forehead-flap reconstruction of a nose completely destroyed by lupus and the correction of ectropion of the right lower eyelid caused by healed lupus scarring of the cheek.

The records show the original condition and the gratifying appearance at the time of the patient's return to Dublin five weeks later. She will return later for a cartilage graft to the nose which will give greater definition to the bridge line. The ectropion has been completely corrected and the extent of the graft is clearly seen in both front and side views.

Case XI (fig. 6).—A further case of facial burns illustrating the reconstruction of an eyebrow by a free hairy graft.

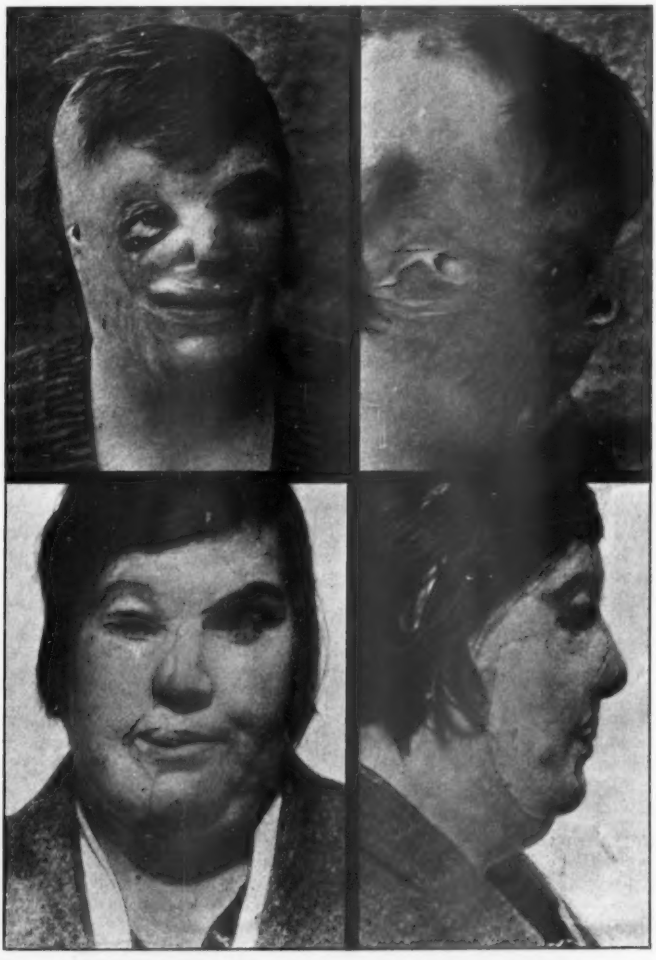


Case IX. FIG. 5.—Severe burn of right cheek dragging eyelid down and lip mucosa up: both deformities treated by Thiersch grafting.



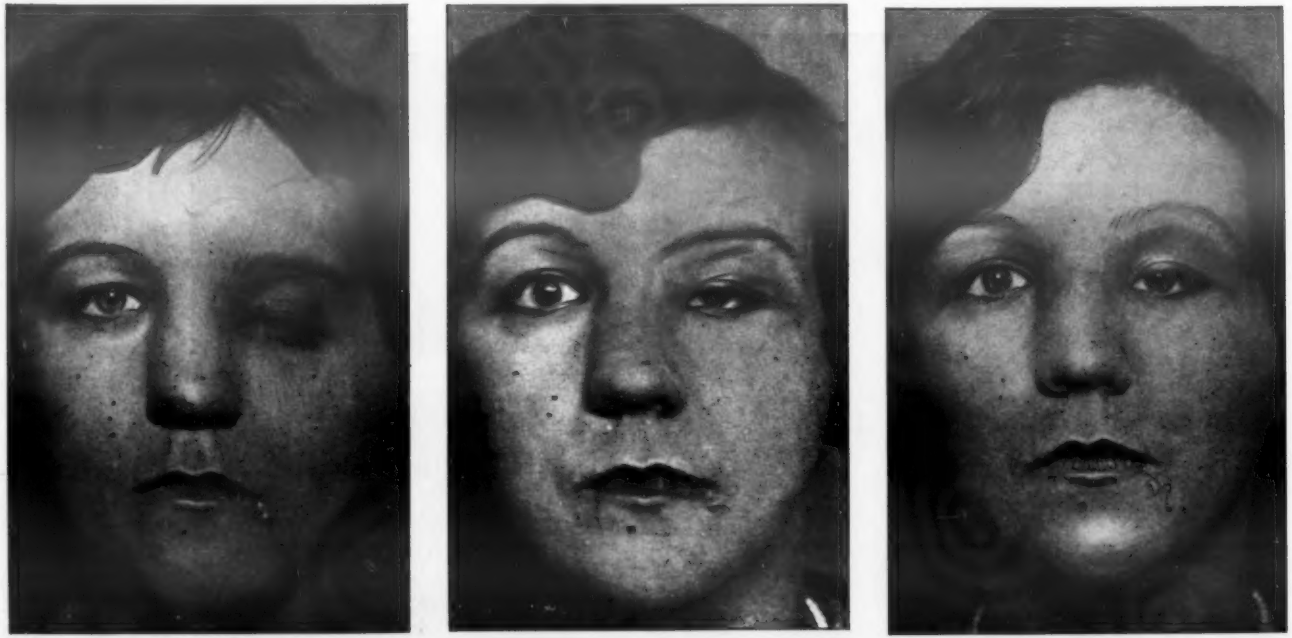
Case XI. FIG. 6.—Extensive facial burn. Ectropion and epicanthus treated by Thiersch grafting: eyebrow restored by hair-bearing Wolfe graft.

Case XII (fig. 7).—A case of severe burns of the face with marked ectropion and loss of part of the nose and the whole of the right ear. The patient was an epileptic and fell on the fire, remaining there for a long time before she was discovered. The cheek and nose have been reconstructed by flaps, and the eyelids have been Thiersch grafted. An eyebrow has been added by the method previously described.



Case XII. FIG. 7.—Severe burn of face. Ectropion treated by Thiersch grafting: eyebrow reconstructed by hair-bearing Wolfe graft; nose and cheek reconstructed by full-thickness skin flaps.

Case XIII (fig. 8).—A case in which extensive loss of bone in the malar and infraorbital regions followed a motoring accident. Diplopia was present and eyelids and eyebrow drooped considerably. There were ugly depressed scars which were excised and a large fat graft from the abdominal wall was inserted to restore contour. Some of the fat was insinuated under the globe.



(A) (B) (C)

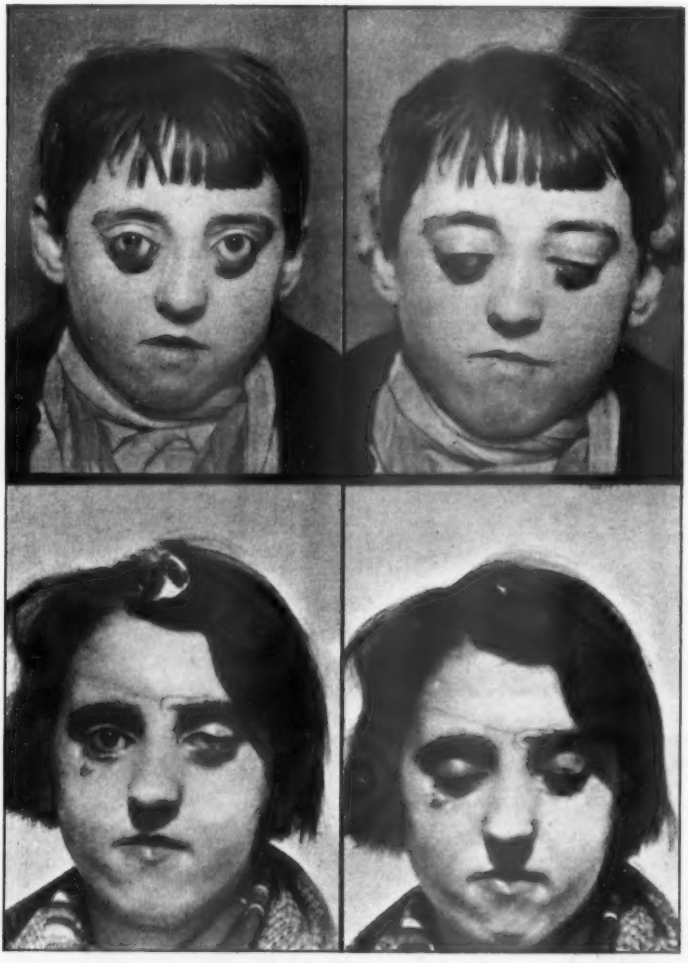
Case XIII. FIG. 8.—(A) Extensive loss of bone in left malar and infraorbital regions. Diplopia was present; and the eye, eyelids and eyebrow drooped considerably. Nasal bridge deflected to right.

(B) Large abdominal wall fat graft inserted to restore malar and infraorbital contour. Part of graft insinuated under globe.

(C) Eyebrow and eyelid raised by elliptical excision in hair-line. Nasal bridge straightened.

The eyebrow was drawn up by an excision in the hair-line. The records indicate that the pupils are now level and the eyebrow and eyelids almost normal. All diplopia has disappeared.

Case XIV (fig. 9).—An unusual case of ectropion, referred by Dr. Gray Clegg, of



Case XIV. FIG. 9.—An unusual case of ectropion following extensive infraorbital necrosis in infancy.

Depressed scars raised by fat grafts: skin loss replaced by Thiersch grafts.

To prevent early contraction of grafts, the eyelids were sutured together after marginal freshening: the lower photographs show the condition before division of the marginal adhesions on the left side.

Manchester, in which extensive destruction of the infraorbital margins followed some apparently trivial injury in early childhood. The history is not clear as to the nature of the bone necrosis, but as it is stated that some bone was discharged from the mouth it is surmised that there might be some luetic factor in the case.

The lower half of the globe was exposed on both eyes. There were deeply depressed scars dragging the lower eyelids down and back to the remains of the orbital floor and completely everting the conjunctiva.

The scarred areas were undermined and fat grafts inserted to fill the depressions. This gave great functional and cosmetic improvement, but it was obvious that there had been some loss of skin. This was made good by Thiersch grafts, and the photographs indicate the result obtained to date.

Case XV.—This is a case of very extensive lupus of the whole face and much of the body, referred to me at the Lord Mayor Treloar Hospital at Alton by Sir Henry Gauvain. Early photographs indicate the desperate condition of the patient before he received treatment by Finsen light. When I first saw him great improvement had been obtained, but there remained complete loss of the nose with stenosis of the nasal airways, eyelids partly destroyed and no longer capable of covering the eyes and extensive corneal opacities.

Mr. Duke-Elder saw him and agreed that every effort should be made to secure a covering for the eyes, as the patient had some sight remaining. In spite of the still active patches of lupus about the face, I was fortunate enough to succeed in providing covering for both eyes by means of Thiersch grafts, with the addition of a small forehead flap on the left side.

The upper lip and nose have been reconstructed by a tubed-pedicle flap brought up, via the wrist, and it will be seen that sufficient material is now in place to transform a hideous-looking object into a presentable being.

Discussion.—Mr. O. GAYER MORGAN asked as to the secretion from the socket. Some sockets, when they had been grafted, became unpleasant; there was an unpleasant odour from the sodden epithelium, especially when the glass eye was removed. He would like to know whether Mr. Kilner used any method of scraping away the sodden epithelium as it accumulated.

Mr. W. S. DUKE-ELDER said that from time to time he had seen at the Hospital at Alton the last case described, and he could confirm what Mr. Kilner said about the terrible appearance. It was difficult to see where the cornea began and where the sclera began; the whole eye was a mass of grey tissue, and it was difficult to know how much was lupus, and how much was the result of exposure. But since the lids had been partially formed, the cornea had cleared up a good deal. It was extraordinary how quickly the vision came back.

In fitting artificial eyes the main deformity which worried people was, in many instances, the hollowness in the upper lid above the artificial eye. He would like to know whether there was any simple way of giving a normal full contour to the upper lid in those cases; it would greatly help the appearance.

Mr. F. A. WILLIAMSON-NOBLE said, with regard to cases in which there were moderate degrees of ectropion of the lower lid, that he had received a small reprint from Dr. Wheeler, of New York, in which it was suggested that if one could take some of the skin of the upper lid where it was loose and put it into the lower lid it would be an advantage, as it was the same colour, and it could be removed without causing subsequent deformity, also it was difficult to detect the junction of old and new.

Mr. J. FOSTER asked whether there was any method by which one could replace lost conjunctiva when the eye was still present. Most of the plastic operations in this region had as their object re-forming a socket in which the eye had been destroyed, and to retain a prosthesis. Some attempts had been made with mucous membrane from the mouth to replace conjunctiva, but it contracted so much that it was difficult to retain it in position. Attempts to re-line an eye socket with Thiersch grafts involved so much irritation to the cornea that it was impossible to retain the eye or the graft. Had Mr. Kilner been able to get over that difficulty?

Mr. KILNER (in reply) said that he occasionally encountered the unpleasantly smelling socket. It was due, he thought, to the exfoliation and maceration of epithelium from the

graft. He cleaned out the socket with pledgets of cotton-wool soaked in peroxide and trained the patient to do this for himself. He had had no case in which the smell persisted after the graft had become finally established. Grease massage to the graft, carried out by the patient himself, helped to prevent contraction in the early post-operative period.

He always used a mould much larger than the final prosthesis, because allowance must be made for contraction, and his experience of skin-grafting in the mouth made him stress the necessity for *excising* all scar tissue and not merely attempting to epithelialize chink-like sulci produced by simple *incision*.

As was illustrated by cases at Sidcup, newly established sulci, whether in mouth or eye-socket, would contract in a remarkably short time. The temporary prosthesis should not be left out of position for longer than was required to carry out the essential toilet of the new cavity.

He agreed that it was unnecessary to disturb any normal mucous membrane remaining on the inner aspect of the eyelids but he did not see why a Thiersch graft in this situation should lead to a thickened appearance of the lids. The socket case he had demonstrated did not show this defect, although both eyelids were covered by graft on their inner surfaces. He imagined that the thickness referred to was due to the use of either a whole thickness skin-graft or a very thick Thiersch graft. The thinnest possible graft should be employed: the use of a Wolfe graft, apart from its thickness, gave the risk of troublesome growth of hair in the socket.

Reference had been made to the question of hollowness in the upper eyelid region. He depended on the artificial eye maker for the prevention of this by the shape of the eye. Fat could readily be introduced in this region, but he did not think that to do this would be tackling the problem correctly. After all, the globe was responsible for the normal protrusion of the upper eyelid and the artificial eye fitted into the grafted socket should be capable of imitating the globe.

Mr. Duke-Elder had referred to the Alton case. He hoped that Mr. Tudor Thomas might be persuaded to carry out a corneal graft on this patient: it would be a great achievement to make the boy see reasonably well now that he was beginning to have passable looks.

Wheeler, of New York, was a master of eyelid reconstruction and there was no question that if normal eyelid skin could be obtained for the repair of an eyelid the best cosmetic result would be obtained. In none of the cases shown had there been available any upper eyelid skin for use on the lower lid. This absence of local material was common to most burn cases. He (Mr. Kilner) sometimes used the skin of the upper eyelid as a double-pedicled flap (Tripier method) in the reconstruction of a lower lid.

Reference had been made to mucous membrane grafting into ocular sulci when the eye was still present. Sir Harold Gillies and he had carried out a number of such grafts and had reported the results in a short paper.[1] The difficulty in these cases was to ensure proper pressure dressing of the grafts and they had been greatly helped in this respect by Mr. Millauro, who had expended much time and skill in preparing shells which could be worn in front of the eye without producing cupping effects on the cornea. These shells, alone or with the addition of small wax moulds, kept the grafts pressed home on the raw surfaces produced by division of adhesions between eyelid and globe. In some cases it had been possible to hide a disfigured but movable eye by a permanent shell-prosthesis of this kind.

There were two further conditions which he would have liked to talk about—ptosis and facial paralysis. He did not know the best procedure for the former, but had obtained gratifying results by following Blaskovics' technique and approaching and shortening the levator from the inner aspect of the eyelid. He felt that this was perhaps trespassing on the ophthalmic surgeon's field.

For facial paralysis he and his colleagues had been using fascia lata grafts with increasingly satisfactory results. He had a case now in a nursing home in which, in addition to the sling-grafts to support the lower face, he had introduced a fascia lata strip from the temporal region through the tissues of the upper eyelid, anchored it around the internal palpebral ligament and carried it back to the temporal region through the tissues of the lower eyelid. The result of this procedure, originally described by Blair and Ferris Smith in America, was most encouraging.

1 H. D. Gillies and T. P. Kilner, "Symblepharon: Its Treatment by Thiersch and Mucous Membrane Grafting," *Trans. Ophth. Soc.*, 1929, xliv, 470-479.

Section of Orthopædics.

President—HARRY PLATT, M.D., M.S., F.R.C.S.

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PRESIDENT'S ADDRESS.

Some Observations on Bone Tumours.

By HARRY PLATT, M.D., M.S., F.R.C.S.

Introduction.

UNDER the conditions of hospital organization in this country, the bone tumours which, year by year, fall to the lot of the individual surgeon are few in number. In this generation, however, we are fortunate in being able to draw on the vast collective experience represented in two famous repositories of bone tumours: the Bone Sarcoma Registry of the American College of Surgeons, founded by Codman, and the Bloodgood Collection at the Johns Hopkins Hospital. These two sources have inspired some of the outstanding contributions to the literature of recent years, and much of what I have to say in this paper has been gleaned more especially from the writings of American surgical pathologists.

For my present purposes I have selected from my own limited material of about one hundred bone tumours—accumulated, with few exceptions, during the past twelve years—a number of tumours to serve as a commentary on the clinical picture of three types of bone tumour: the *chondroma*, the *giant-cell tumour* and the *sarcoma*.

The Chondroma.

The term "chondroma" should properly be reserved for tumours which remain predominantly cartilaginous throughout their life history. This definition excludes the exostosis (or osteochondroma) in its primitive form, but not the tumour which results from the proliferation of the cartilaginous cap of an exostosis, and which is indistinguishable from the pure chondroma.

The chondroma may be represented by two main clinical types: (I) the solitary chondroma of the long bones and flat bones, (II) the enchondroma, single or multiple, of the digital bones of the hand and foot. Both types originate in childhood, are generally discovered before the end of the second decade, but not infrequently escape recognition until a later stage.

(I) *The Solitary Chondroma: General characteristics.*—The favourite sites for the solitary chondroma are (1) the ends of the major long bones: femur, tibia (especially the region of the knee), and humerus; and (2) the pelvis. Although these tumours may be classified as "periosteal" growths, it must be realized that the chondroma tissue often penetrates for some distance into the interior of the affected bone. This is an important observation in relation to the effective excision of the tumours and their liability to recur locally.

The clinical picture of the solitary chondroma is familiar to all surgeons: the small tumour, unsuspected in the early stage; the absence of symptoms for some years; and later the appearance of a tumour large enough to be palpable or visible. Some tumours grow slowly throughout their course; others grow rapidly from the beginning.

The radiographic picture of the large chondroma is characteristic (fig. 1); a dense shadow with a feathery outline, composed of deeply calcified "splashes." The contrast in density between the tumour shadow and the adjoining bone is striking. Very rarely the tumour is completely invisible. (Cases 4 and 5.)

To the naked eye the chondroma appears as a well encapsuled, lobulated mass of semi-translucent cartilage. The bulk of the tumour shows a uniform histology—adult cartilage, mixed with embryonic cartilage, and here and there the stellate cell areas of the myxoma pattern. Small patches of ossification are also common, but conversion into bone on a large scale rarely, if ever, occurs. (See Case 1.)

Special clinical features.—The solitary chondroma assumes special interest and importance to the surgeon, when a tumour (*1*) attains to relatively enormous dimensions; (*2*) becomes locally invasive and destructive; or (*3*) undergoes malignant transformation.

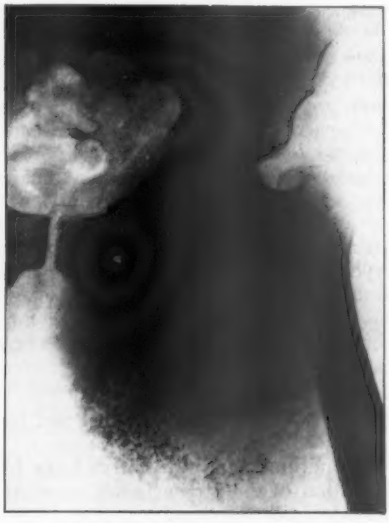


FIG. 1.—*Case* 2. Large chondroma arising from the front of pubis. Note the feathery outline and the patches of dense calcification.

(*1*) *The large tumour.*—A solitary chondroma, hitherto small and quiescent, may show renewed activity at almost any age-period—childhood, adolescence, middle age, or even advanced life.

It is important to realize that the onset of rapid growth and the attainment of enormous size may be seen in encapsuled tumours which retain their benign qualities. Rapid growth and large size, *per se*, are not pathognomonic of the advent of malignancy.

In certain regions there are obvious risks attached to the presence of a large growing tumour. But on the whole pressure signs are late phenomena, even in gigantic growths. The significance of rapid growth and unusual size is illustrated in the following tumours.

Case 1.—1922. E. R., male, aged 59. Chondroma of Neck of Femur (L). Sciatic Nerve Involvement.

History.—Gradually increasing sciatica for ten years.

On examination.—Limitation of movement in the left hip; hard tumour, size of a fist, in gluteal region; firmly fixed to femur. No sign of loss of conduction in sciatic nerve.

X-ray.—Large tumour covering posterior aspect of femoral neck and upper part of shaft; dense lobular calcification. (Fig. 2.)

Treatment. — (20.1.22.) *Excision of tumour; cauterization of tumour bed.* The tumour was removed without difficulty after freeing the sciatic nerve from a gutter on the posterior surface of the tumour mass. The tumour consisted of an encapsuled mass of cartilage and bone.

Histology.—Chondroma showing extensive areas of ossification.

Result.—Five years later—no sign of recurrence.

Comments.—This case illustrates: (i) the attainment of large size by comparatively slow growth; (ii) pressure on an important nerve trunk; and (iii) an unusual amount of ossification. The tumour more properly belongs to the osteo-chondroma group.

FIG. 2.—*Case* 1. Large chondroma arising from the posterior aspect of neck of femur.

Case 2.—1931. J. F., male, aged 34. Chondroma of Pelvis.

History.—A lump first noticed in the left groin twelve months ago; has grown slowly until recently; no pain; no interference with function of the lower limb.

On examination.—A large mass in Scarpa's triangle, stony hard and painless, the size of a fœtal head, fixed to the pubis and ischium. Full mobility of the hip-joint.

X-ray.—Large tumour with patchy calcification, arising from the front of the pelvis on the left side. (Fig. 1).

Treatment.—(27.2.31.) *Excision of tumour; curettage of tumour bed.* Exposure was difficult, owing to the enormous size of the tumour which was attached by a narrow pedicle to the front of the pubis and ischium. The chondroma tissue extended deeply into the symphysis, the interior of which appeared to have been almost completely transformed into friable cartilage.

Description of tumour.—An encapsuled mass of cartilage lobules.

Histology.—Cartilage cells, chiefly of adult type; no myxoma areas discovered.

Result.—No sign of recurrence up to date (eight months).

Comments.—A rapidly growing chondroma of enormous size, but with no evidence of malignant change.

Case 3.—1922. C. W., male, aged 19. Multiple Exostoses. Diaphyseal Aclasis. Large Chondroma of Tibia.

History.—Known to have multiple exostoses for many years (father similarly affected). During past two months a small tumour in the upper part of the right tibia has grown rapidly and attained the size of a football.

On examination.—Large tumour involving the upper end of the right tibia, and occupying the whole circumference of the limb; hard and painless with small softened areas (fig. 3).

X-ray.—Typical chondroma shadow (other bones show small multiple exostoses with the appearance of diaphyseal aclasis) (fig. 4).

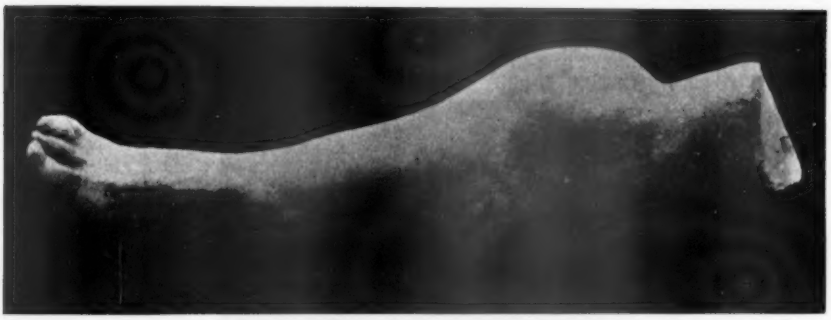


FIG. 3.—*Case* 3. Cast of amputation specimen showing large chondroma arising from the upper end of tibia.

FIG. 4.—*Case* 3. Radiogram of tumour shown in fig. 3.

Treatment.—(4.8.22.) *Amputation of leg* (*L.*)—After an unsuccessful attempt to excise the tumour, the limb was amputated through the lower third of the thigh.

Description of tumour.—Lobulated masses of pure cartilage completely encapsulated.

Histology.—Cartilage tissue, with myxomatous areas; very cellular picture with a suggestion of mitoses.

Result.—Alive and well nine years later. No appreciable increase in size in other tumours.

Comments.—(i) A chondroma originating in the cartilaginous cap of an exostosis in a case of diaphyseal aclasis. (ii) Rapid growth and the attainment of large size. (iii) Tumour invading the interior of the shaft, but no clinical or histological signs of malignancy.

(2) *Local invasion and destruction.*—Although the stalk of a chondroma often penetrates into the interior of the affected bone, the average tumour increases in size without in any way weakening the bone at the site of its origin. Occasionally, however, a chondroma may become invasive and destructive, but without the loss of its benign characters. In such circumstances spontaneous fracture may occur—a most unusual phenomenon in the career of the solitary chondroma. An example of this uncommon complication may be recorded here.

Case 4.—1927. J. R., male, aged 50. Chondroma of Neck of Femur (L). Spontaneous Fracture.

History.—A few months of intermittent limp with pain referred to the left knee, followed by sudden collapse of the left leg when walking; no antecedent violence.

On examination.—Typical clinical signs of fracture of the neck of the left femur.

X-ray.—Shows fracture of the femoral neck, with no signs of gross bony destruction, and no tumour shadow (fig. 5). A careful clinical examination revealed no evidence of primary growth in any part of the body.

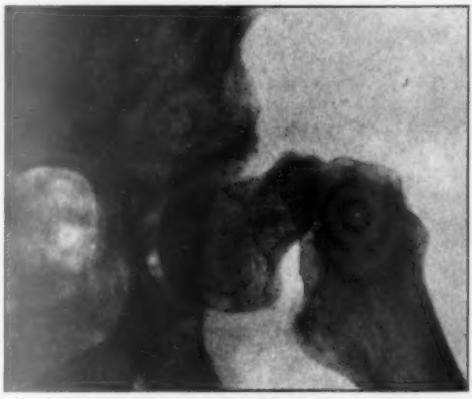


FIG. 5.—*Case 4.* Spontaneous fracture through base of neck of femur. Note the absence of gross bony destruction and tumour shadow.

Treatment.—(21.6.27). *Excision of head and neck of femur with attached chondroma.* On opening the hip-joint capsule, the femoral neck was seen to be encircled by a lobulated tumour imperfectly encapsuled. An attempt was made to excise the cartilaginous masses, which were unusually friable; this proved impossible, and it was found necessary to resect the neck and head of the femur *en masse* with the remains of the tumour.

Description of tumour.—Clusters of cartilage, creamy white, very friable, encircling the femoral neck; localized erosion of the margins of the head and upper border of the neck (fig. 6).

Histology.—Typical chondroma.

Result.—Four years later—no sign of recurrence. The patient is well satisfied with his somewhat crude pseudarthrosis of the hip.

Comments.—A comparatively small chondroma in a man of 50, producing destruction of the femoral neck, resulting in spontaneous fracture. No evidence of malignant transformation.

(3) *Malignant transformation.*—It is generally taught that a small proportion of innocent tumours of the fibro-cartilaginous group ultimately become malignant. But many of the examples of malignant degeneration in a chondroma or exostosis recorded in surgical literature, lack convincing histological proof. It is obvious that the presumed malignant change has been simply the occurrence of unusually

rapid growth in a benign encapsuled tumour. More than seventy years ago, Sir James Paget [1] gave a striking description of the neglected cartilaginous tumours, which grow to gigantic size, and, as he said, "imitated the progress of malignant disease."

There is, however, ample evidence that an osteogenic sarcoma may develop at the site of a chondroma or exostosis. The American Sarcoma Registry includes undoubted specimens verified by Kolodny [2], Phemister [3] and others. The Johns Hopkins collection also contains a group of tumours—formerly regarded by Bloodgood as the rare myxoma of bone, which are now believed to be chondro-sarcomata arising in pre-existing benign fibro-cartilaginous growths. (Geschickter [4].) The study of these "sequential" tumours has emphasized the difficulty of establishing the histological criteria of malignancy. It is also to be noted that the tumours are extremely radio-sensitive, and after local excision or amputation provide an unusually high percentage of five-year cures.

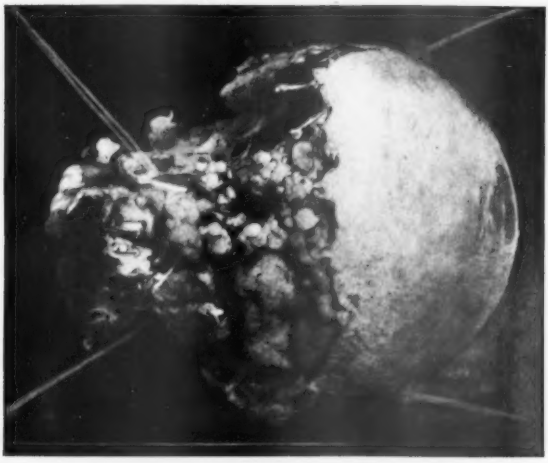


FIG. 6.—*Case* 4.—Specimen after excision, showing clusters of cartilage encircling the femoral neck.

True malignant transformation is usually seen after the age of 35, and is heralded by a rapid increase in the size of the tumour, which, sooner or later, shows the unmistakable clinical signs of a sarcoma of bone. The following example of a gigantic chondroma is perhaps rather suggestive of malignant transformation, but the final proof afforded by autopsy is wanting.

Case 5.—1928. C.B., male, aged 70. Chondroma of Ilium (L).

History.—Pain on anterior aspect of left thigh for eighteen months, occasionally radiating down to the ankle. Ten days before reporting at hospital swelling was discovered above the left hip.

On examination.—Decrepit looking individual. A large globular tumour involving the posterior part of the left ilium, extending into the iliac fossa and outwards into the buttock. Tumour firm, but not stony hard; painless to touch; no objective signs of pressure on any important structure. Movements of the hip-joint free. No clinical or radiological evidence of primary or secondary growths elsewhere. Wassermann test, negative.

X-ray.—Some irregularity at the lower margin of the left sacro-iliac joint. No evidence of gross destruction or expansion of ilium; no extra-osseous shadow.

Treatment.—(1) 15.6.26. *Exploration of tumour.* The gluteus maximus was split and the tumour capsule exposed. A small portion of typical chondromatous tissue was evacuated. Rapid histological section showed the structure of a chondroma. The tumour was regarded as benign, but owing to its size and the general condition of the patient, complete excision was impracticable. Wound closed and arrangements made for radium treatment.

(2) 22.6.28. *Re-exploration of tumour. Insertion of radium needles.* In order to expose a large part of the tumour the muscular origins were stripped from the iliac crest. Eight radium needles were inserted at various levels into the tumour mass.

Subsequent progress.—Some sloughing of the wound followed and healing was slow. Six months later a large tumour was easily palpable in the buttock and iliac fossa; the patient's general condition had in no way deteriorated.

Result.—Death took place one year and ten months from the date of the operation. The tumour had increased to a tremendous size, but no information regarding possible malignancy was available.

Comments.—An enormous chondroma of the ilium in a man of 70, with a short clinical history. Tumour encapsuled and on histological evidence benign.

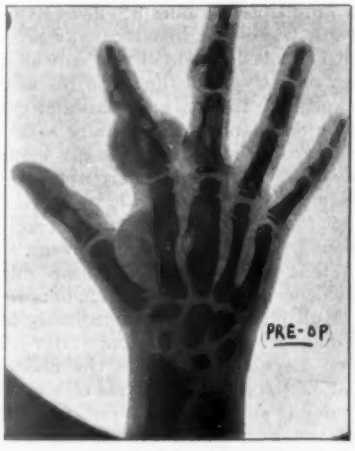


FIG. 7.—Multiple chondromata of digital bones in a boy of eight years. Note (i) extensive changes in several bones; (ii) the solid tumours.

(II) *Enchondroma* (*Myxochondroma*).—We may now glance at the chondroma in its miniature guise, as a tumour of "central" origin, with a special predilection for the long bones of the hand and foot. This almost exclusive skeletal distribution is well exemplified in the Johns Hopkins material, in which out of 70 central chondromata, three only involved the major long bones (femur, 2; tibia, 1).

The digital enchondroma originates in early childhood, in the growing end of a phalanx, a metacarpal, or less commonly a metatarsal. In the course of its evolution a tumour may give rise to a variety of changes: (1) The interior of the shaft may be invaded until it becomes almost completely occupied by tumour growth. A bone so transformed may retain its cartilaginous structure for years, but in later life a considerable degree of spontaneous reconstruction may be seen. (2) A solid cartilaginous nodule may push its way to the surface and form a localized tumour, especially appreciable on clinical examination (fig. 7). (3) The chondroma may be converted into a miniature bone cyst.

These various phases may be followed in radiograms of multiple chondromata, in which the typical combination of solid tumours and chondromatous transformation of a number of digital bones may be demonstrable at the same epoch.

Two clinical types of digital chondroma may be conveniently recognized: (*a*) the solid tumour; and (*b*) the chondromatous cyst.

(*a*) *Solid tumours.*—Solid chondromata occur as either single or multiple tumours. When bilateral their distribution is usually asymmetrical. Like multiple exostoses, to which they are closely allied, multiple enchondromata represent a special type of hereditary growth disturbance. The majority of the tumours become apparent under the age of ten, and if ignored may grow to enormous dimensions.

Operative treatment is indicated in rapidly growing tumours which have become unsightly, and a source of inconvenience. Complete excision, with curettage and cauterization of the tumour bed, is essential, as there is an undoubted tendency to local recurrence. When a phalanx bearing a solid tumour has been largely converted into chondromatous tissue, effective curettage may lead to the collapse of an incomplete and fragile bony shell. In such circumstances the shell should be packed with autogenous bone chips, and the digit carefully splinted. For mechanical reasons it is wise to remove multiple tumours at successive operations.

(*b*) *The chondromatous cyst* (*myxochondroma*): *Clinical and pathological picture.*—I shall deal very briefly with this lesion, which was considered in some detail, nearly two years ago, in my paper on "Cysts of the Long Bones of the Hands and Feet" [5]. It will suffice to recapitulate the main clinical and pathological facts.

The cysts are more commonly single, and usually arise in the metaphysis of a proximal phalanx or metacarpal, the favourite digit being the little finger. The majority of chondroma cysts develop insidiously, remain symptomless for a long time, and are revealed accidentally in a radiogram obtained after some comparatively minor injury to the hand or foot. During the stage of active expansion, when the cyst wall is thin, spontaneous fracture is a common event.

I have already shown that a proportion of these miniature cysts which, on clinical and radiological evidence, might pass as enchondromata, actually belong to the *ostitis fibrosa* group. Multiple-lesions are more likely to be chondromata but in single cysts, a differential diagnosis is possible only after a histological examination of the cyst contents. For diagnostic purposes the chondroma and ostitis fibrosa embrace the whole morbid histology of the cysts of the digital bones, a region in which the true giant-cell tumour and osteogenic sarcoma are almost unknown.

The special interest of the chondroma cyst lies in its microscopic anatomy, in which is displayed, par excellence, the active cellular picture of the myxochondroma, a tumour to be regarded with suspicion when appearing as a central tumour of a major long bone. In the miniature bones, however, the myxochondroma behaves in every respect as a benign lesion.

Treatment.—It has been my practice in the past to explore all digital bone cysts discovered during the stage of active expansion, or after the occurrence of spontaneous fracture. The rationale of the operation is based on the rapid and permanent healing of the cyst, which follows effective curettage, cauterization, and the insertion of bone grafts. Thick-walled cysts in a state of quiescence should be left untouched.

The Giant-cell Tumour or Osteoclastoma.

Of the several titles under which this tumour is known, perhaps the most appropriate is the one in current use in America—*benign giant-cell tumour*. Although, in some respects, this title is non-committal, it is, in my view, an apt description of a lesion which represents a clinical rather than a histological entity. The alternative titles, "myeloma" and "myeloid sarcoma," are inaccurate and misleading: myeloma, because it signifies a tumour of specific narrow cells, as in the rare but well recognized affection, "multiple myelomatosis"; and myeloid sarcoma,

because it tends to attribute a definite malignant significance to a tumour which beyond all doubt is fundamentally innocent.

Clinical picture: *Incidence.*—One of the surprising facts relating to the incidence of bone tumours in general is the comparative infrequency of the giant-cell tumour. This statement is true only when applied to the *endosteal* tumour of the long bones, spine, or pelvis. If the familiar *epulis* of the jaw is included, a different impression will be formed. There is, however, no logical reason why this *periosteal* lesion, despite its histological characteristics, should be grouped with the giant-cell tumour proper.

The undoubted rarity of the giant-cell tumour is borne out in most hospital statistics. From figures collected some years ago by Stewart [6], the average quota of giant-cell tumours of the long bones in some of the larger hospitals is one per year per hospital. In my own clinics, during a period which has provided me with twenty-four cases of bone sarcoma and twenty solitary metastatic growths, twelve giant-cell tumours only have fallen to my lot.

The giant-cell tumour as a clinical entity.—I have referred to the giant-cell tumour as a clinical entity. This conception is essential to the practical surgeon, for it allows us to place the tumour in its correct "setting" and to define with some certainty the principles which should guide us in prognosis and treatment.

The broad outlines of the picture of the giant-cell tumour can best be displayed when the tumour is ranged alongside the solitary bone cyst on the one hand, and the true sarcoma of bone on the other. The main points of resemblance and contrast are set forth in Table I (p. 10).

From this comparison, the giant-cell tumour emerges as a clear-cut entity; we note its distinctive age and sex incidence, skeletal distribution, site of origin, sequence of clinical events, and the behaviour of the tumour if its progress is unchecked by treatment. But the tumour tends to lose something of its identity when its histogenesis is contemplated. Here we see the suggestion of a possible relationship between lesions which otherwise are in striking contrast.

Relationship of the giant-cell tumour to the benign bone cyst.—It has been known for some time to surgical pathologists that the bone cyst and the giant-cell tumour are linked together by a common histological denominator—*the giant-cell area.* This fact, foreshadowed by von Recklinghausen in his original description of ostitis fibrosa, has led to much confusion in the diagnosis and classification of cystic affections of bone. Its influence can be traced in the earlier beliefs (*a*) that the giant-cell tumour was a common affection; (*b*) that its diagnosis rested essentially on histological findings without reference to any special combinations of clinical factors. We must now recognize that the majority of the giant-cell tumours hitherto described under the age of 15, have either been cysts, or, in exceptional cases, true sarcomata with an atypical histology.

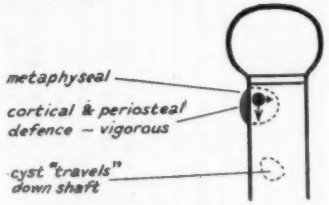
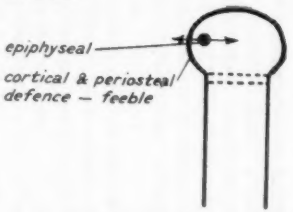
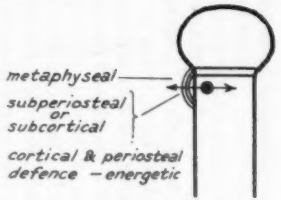
Some of the giant-cell areas discovered in bone cysts present features which do not always conform to the typical giant-cell tumour histology. This difference is chiefly concerned with the number and size of the giant-cells—which in ostitis fibrosa are often scanty, small, and contain relatively few nuclei.

Between the ages of fifteen and twenty-five—the border-land period—when the cyst is fading out of the picture and the giant-cell tumour is just appearing, there may be some little difficulty in distinguishing between the two lesions. This is most likely to happen in cysts in certain situations discovered about the age of twenty, in which the histology approximates to the typical giant-cell tumour of older individuals; i.e., abundant, large-sized giant cells, with numerous nuclei. In the last analysis, however, it is the clinical syndrome which counts, although on occasion the final diagnosis may be retrospective.

In this connection, the following cases from my personal series are instructive:—

Case 6.—A. F., female, aged 17. Cyst of Great Trochanter (L). ? Giant-cell Tumour: ? Ostitis Fibrosa.

TABLE I.

	Benign Bone Cyst.	Giant Cell Tumour.	Osteogenic Sarcoma.
Origin and mode of extension	*Age* 5—15 Under 20 *Sex* M. > F.	*Age* 20—35 Over 20 *Sex* F. > M.	*Age* 10—40 *Sex* M. > F.
			
	X-ray.—Symmetrical expansion, trabeculation faint or absent	*X-ray.*—Multicystic expansion—eccentric. Trabeculation marked and coarse. Perforation of cortex	*X-ray.*—Spindle swelling Erosion of cortex Radiating ossification (18%) Extra-osseous tumour shadow
Site of election	Humerus ... Upper end Femur ... Trochanteric region Tibia ... Upper end	Femur ... Lower end Tibia ... Upper end Radius ... Lower end	Femur ... Lower end Tibia ... ... Upper end Humerus ... Upper end Pelvis
Clinical course	Trauma → fracture (45%)	Trauma → pain → tumour ↓ (fracture—13%)	Trauma → pain → tumour (fracture—8%)
Morbid anatomy	Cyst { empty — fibrous lining contents { clear fluid mixture of bone and fibrous tissue liquid blood	Reddish-brown tumour tissue—maroon-coloured areas—liquid blood	Tumour tissue—friable ; reddish-brown. Areas of myxoma, cartilage, ossification
Histology	Spindle cells and fibroblasts Osteoid bone Hæmorrhages (Myxoma and cartilage) Giant cell areas { typical atypical	Round cells Spindle cells No ossification from tumour cells Giant cells { abundant large multinucleated	Sarcoma cells—oat-shaped—small and large (mitoses) Osteoid bone Myxoma and cartilage Ossification from tumour cells Malignant giant cells { few small nuclei few
Pathogenesis	Non-tumour lesion—a tissue reaction Spontaneous healing + + +	Benign neoplasm Invasive and destructive	Malignant neoplasm Destructive and metastatic

History.—Limp of few weeks' duration.

On examination.—(10.10.22.) Left trochanter prominent and tender; slight limitation of abduction and internal rotation of hip.

X-ray.—Shows cystic expansion outer part of great trochanter; coarse trabeculation; cortex thin (fig. 8).

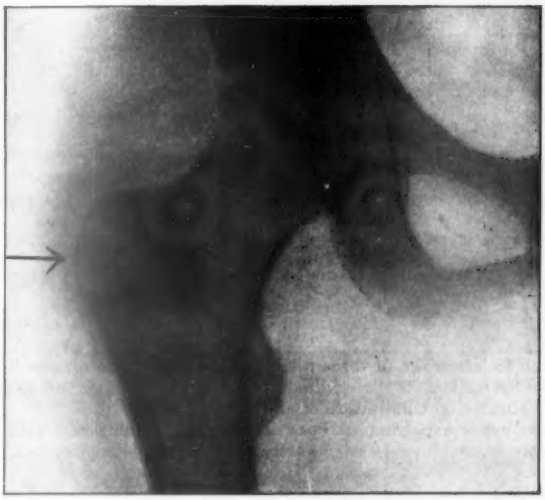


FIG. 8.—*Case* 6. Trabeculated cyst in the great trochanter with thin cortex.

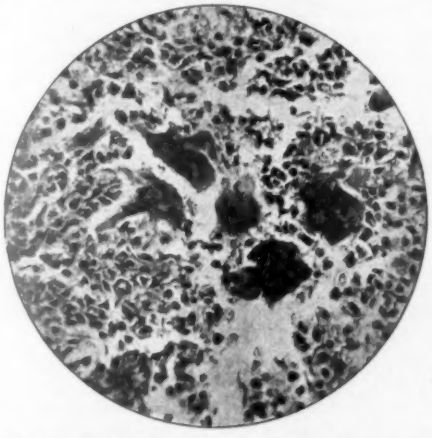


FIG. 9.—*Case* 6. Cyst of great trochanter (L). ? Giant-cell tumour; ? Ostitis fibrosa. Contents of cyst showing large giant cells of tumour type. High-power.

Treatment.—(1.12.22). *Exploration of cyst: curettage and cauterization.* Small perforation discovered in cortex through which a mass of friable reddish-brown tissue protruded; interior of cyst filled with similar tissue; no definite lining; contents evacuated and cavity cauterized with pure carbolic acid.

Histology.—Very cellular picture—polygonal cells; giant cells large and multinucleated (fig. 9).

Result.—Nine years later, cyst completely obliterated.

Comments.—A cyst in a patient under the age of 20, in which both the macroscopic and microscopic anatomy of the contents suggested giant-cell tumour rather than ostitis fibrosa; but occurring in a site (greater trochanter) known to be common in benign cysts, but rare in giant-cell tumours.

Diagnosis.—Ostitis fibrosa.

Case 7.—E. C., female, aged 15. Cyst of Great Trochanter (R). Ostitis Fibrosa.

History.—Pain and intermittent limp for five months.

On examination.—(25.4.21.) Thickening of right trochanter; slight limitation of abduction and internal rotation.

X-ray.—Shows multicystic expansion of great trochanter; cortex thick.

Treatment.—(30.6.21.) *Exploration cyst; curettage and cauterization.* Cyst occupied by soft greyish tissue; no definite lining; cavity curetted and interior cauterized.

Histology.—Probably ostitis fibrosa; no giant-cell areas.

Result.—Two years later, complete obliteration of cyst.

Comments.—A typical ostitis fibrosa cyst of the great trochanter.

Case 8.—M. R., female, aged 15. Cyst of Pubis (L). ? Ostitis Fibrosa. ? Giant-cell Tumour.

History.—Pain in left groin, of three months' duration; intermittent limp.

On examination.—(9.10.23.) Swelling along brim of pelvis on left side; no fluctuation; no distension of veins: slight limitation of abduction left hip.

X-ray.—Shows cystic expansion of body of pubis on the left side with disappearance of inner part of pubic ramus (fig. 10). Radiograms of other bones: skull, long bones, etc., showed no changes.

Treatment.—(29.10.23.) *Exploration of bone cavity; curettage and cauterization.* The pubis was exposed after detaching the inner end of Poupart's ligament and part of the rectus insertion. An encapsuled tumour was exposed which occupied the remains of the pubis. This capsule formed the wall of a large cavity containing *liquid blood*, and a mass of reddish-brown tumour-like tissue. Marked hæmorrhage was experienced, which necessitated temporary packing. The cavity was completely curetted, and the interior cauterized with pure carbolic acid. The remains of the bone-shell could be felt along the line of the pubic ramus.

Histology.—Very cellular tissue, chiefly consisting of a mixture of spindle and round cells; a few giant-cell areas; giant cells scanty, but fairly large with many nuclei; areas of osteoid bone and hæmorrhages. Preliminary diagnosis: giant-cell tumour.

Result.—Four years later no sign of recurrence; no disability. *X-ray.*—Shows complete re-formation of pubic ramus (fig. 11).

Comments.—(1) A cyst in an unusual situation (pubis) containing liquid blood and tumour-like tissue, suggesting giant-cell tumour, or even sarcoma; (2) a lesion mildly invasive, but cured by curettage and cauterization; (3) on histological grounds, ostitis fibrosa with giant-cell areas conforming to tumour type.

The histological resemblance between the cyst and the giant-cell tumour has tempted certain writers to regard the two lesions as different stages of a single *clinical* entity. The *hæmorrhagic osteomyelitis* hypothesis of Barrie [7], is an example of this illogical reasoning, which ignores the simple fact that the giant-cell tumour always behaves like a true neoplasm. Similar objections may be urged towards the creation of such histological entities as the "giant-cell variant of ostitis fibrosa," or the "spindle-celled variant of the giant-cell tumour"—the hybrid lesions described by Geschickter and Copeland in the digital bone cysts [8]. As I have already pointed out, these lesions have no exact clinical counterpart.

We must hold fast to the essential contrast between the clinical picture of the bone cyst and the giant-cell tumour as the basis of correct prognosis and treatment;

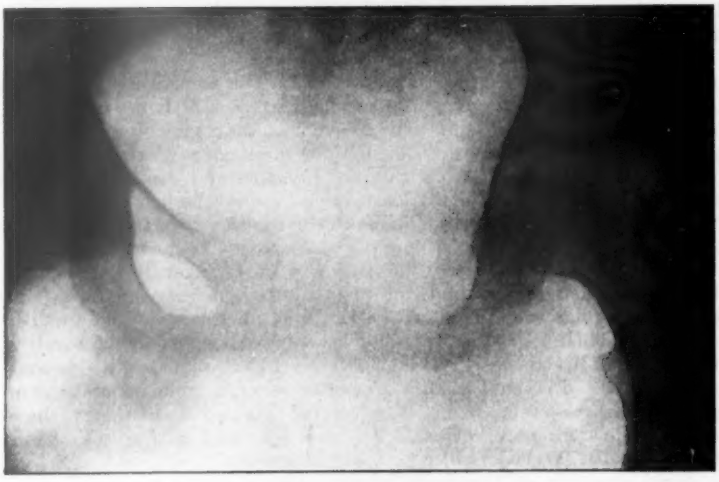


FIG. 10.—*Case* 8. Almost complete disappearance of the pubic ramus and part of pubis; faint trace of bony shell showing.

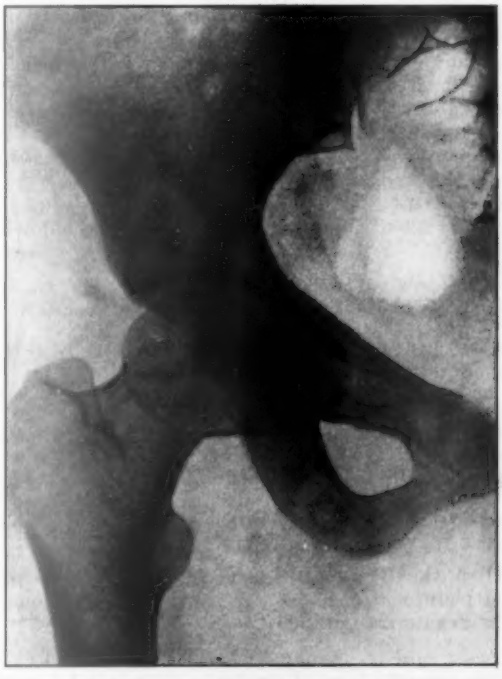


FIG. 11.—*Case* 8. Four years later—shows re-formation of the pubic ramus after operation of curettage.

for, whereas the cyst in certain phases may be treated by operation as a matter of convenience, in the giant-cell tumour complete eradication of the lesion is imperative at the earliest moment.

Relation of the giant-cell tumour to sarcoma.—The recognition of the giant-cell tumour as a benign lesion distinct from the true sarcoma of bone, goes back to the time of Paget and Nélaton. In our generation, this view has been vigorously propagated by Bloodgood, who is responsible for the modern treatment of the tumour by conservative surgical methods. But the invasive and destructive activities of this tumour, and its capacity for local recurrence, cannot be ignored.

From time to time the prevailing belief in the innocency of the giant-cell tumour has been shaken by reports of tumours which have been followed by visceral metastases—usually in the lungs. The majority of the so-called malignant giant-cell tumours recorded in the literature have been true sarcomata in which the histological picture has been misinterpreted. Such mistakes are likely to be rare in the future, as the histological distinctions between the *myeloid* giant cell and the *malignant* giant cell, so clearly enunciated by Stewart (loc. cit.), become more widely appreciated. In the past few years, however, a number of tumours with pulmonary metastases reproducing the typical giant-cell histology, have been described by competent observers and accepted by many authorities. (Finch and Gleave [9], Orr [10], Dyke [11].)

The vexed question of the potential malignancy of the giant-cell tumour has been most ably discussed by Geschickter and Copeland [12] in a critical study of eight so-called metastasizing tumours—three from the Johns Hopkins collection, and the remainder from recent surgical literature. The conclusions of these writers are most interesting, for it would appear that on the rare occasions when a proved giant-cell tumour behaves in malignant fashion, it is not an actual transformation of the original tumour, but the superimposition of an osteogenic sarcoma. This sequence of events is liable to occur only in neglected tumours, or in tumours which have recurred locally after inadequate curettage in individuals over the age of thirty.

Thus, whilst increasing knowledge and experience of the giant-cell tumour reaffirms its fundamental innocency, the necessity for prompt and effective eradication of these growths remains unchallenged.

Prognosis and treatment.—The prognosis and treatment of the giant-cell tumour in various stages of its evolution may be considered in the following cases from my personal series :—

Case 9.—H. R., male, aged 24. Giant-cell Tumour of Femur, Lower End (L).

History.—Pain and swelling left knee, thirteen months' duration: vague recollection of trauma.

On examination.—(7.4.19.) Enlargement of internal condyle femur (L); outward bowing of limb; no interference with function of knee-joint.

X-ray.—Shows multicystic expansion inner femoral condyle with coarse trabeculation; cortex thick (fig. 12).

Treatment.—(15.5.19.) *Curettage and cauterization.* A large mass of reddish-brown tissue was evacuated.

Histology.—Typical giant-cell tumour, with considerable spindle-cell areas and fibrous tissue.

Result.—Twelve years later, no sign of recurrence; cyst almost completely obliterated by new bone (fig. 13).

Comments.—(1) A slowly growing giant-cell tumour, not markedly invasive, with a histological picture in keeping with its clinical behaviour. (2) Permanent cure by curettage and cauterization.

Case 10.—J. M., male, aged 31. Giant-cell Tumour of Ilium (R).

History.—Swelling over right sacro-iliac region, three months' duration; no pain; some stiffness of the lower spine.

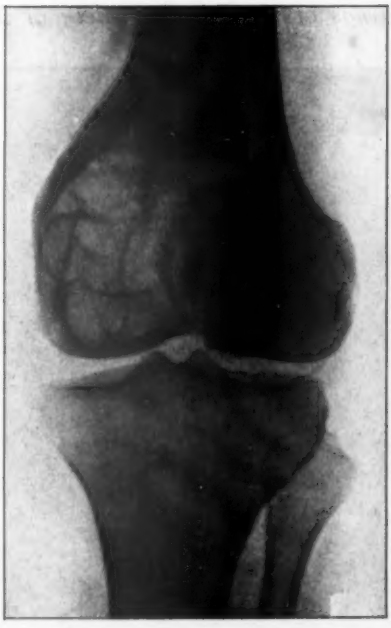


FIG. 12.—*Case* 9. **Multicystic expansion of inner condyle of femur; coarse trabeculation; cortex thick.**

FIG. 13.—*Case* 9. **Condition twelve years later—cystic area almost completely obliterated by new bone.**

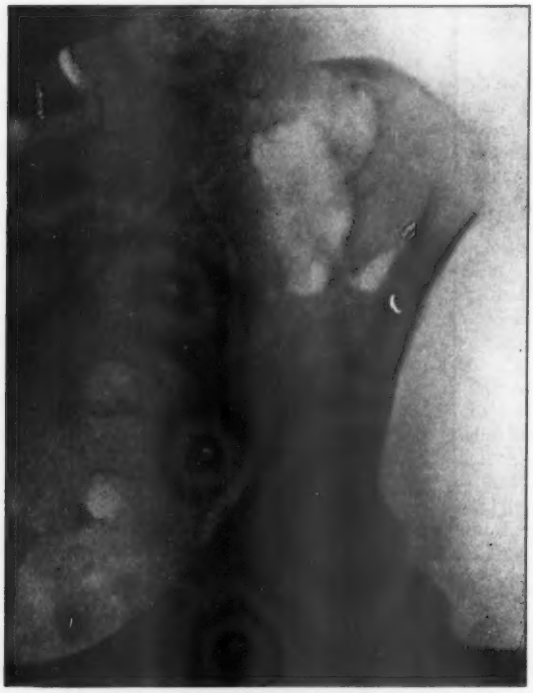


FIG. 14.—*Case* 10. Destruction of the posterior part of the ilium with shadow of tumour above the sacro-iliac joint.

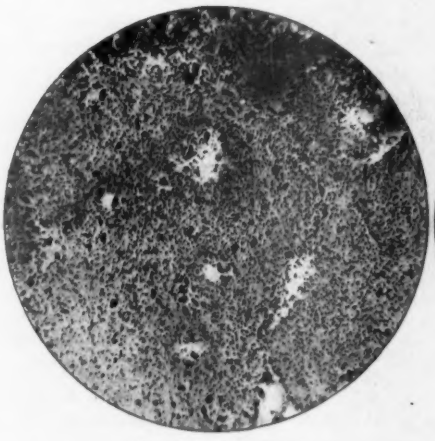


FIG. 15.—Low power.

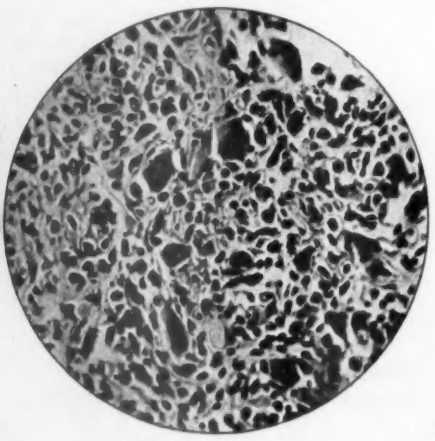


FIG. 16.—High power.

FIGS. 15 and 16.—*Case* 10. Giant-cell tumour of ilium (R). Giant cells fairly abundant, but small, and containing few nuclei.

On examination.—(3.10.24.) Boggy swelling posteriorly over the right sacro-iliac joint.

X-ray—Shows widespread destruction of ilium with shadow of tumour just above right sacro-iliac joint (fig. 14).

Treatment.—(1) (3.12.24.) *Exploration of tumour: curettage and cauterization.* On incising the tumour capsule, very severe hæmorrhage occurred. It was impossible to eradicate all the tumour tissue, and owing to the condition of the patient the operation was suspended and the cavity packed. (2) Three days later *radium* tubes were inserted. Patient discharged from hospital with large granulating cavity in right sacro-iliac region.

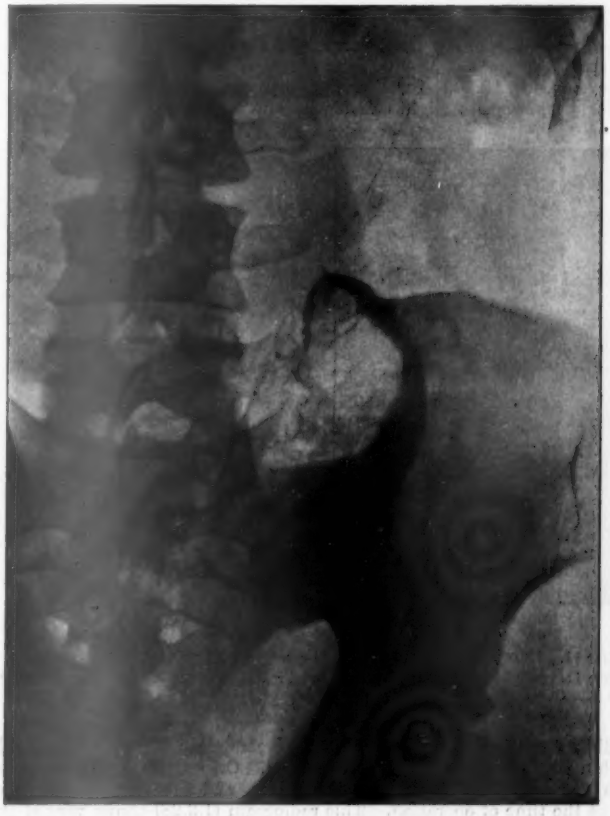


FIG. 17.—*Case* 10. Condition three years later—excavation in the posterior part of the ilium surrounded by an area of sclerosed bone. Appearances suggest a healed lesion.

Histology.—Giant-cell tumour; giant cells abundant but small and containing few nuclei. Preliminary diagnosis at that date by competent authority: Osteogenic sarcoma with malignant giant cells (figs. 15 & 16).

Result.—Seven years later patient still alive, in good health, and following his ordinary occupation. Large depression over the posterior part of the right ilium incompletely lined by skin; at the bottom of this cavity, bone is still exposed and covered by a horny epithelium.

X-ray.—Excavation of ilium—clear-cut outline; obviously healed (fig. 17).

Retrospective diagnosis.—Benign giant-cell tumour.

Comments.—(1) A rapidly-growing tumour of the ileum, markedly invasive and destructive; perforation of bone shell with large tumour mass outside the bone, but still encapsuled. Bone cavity contained liquid blood, and profuse hæmorrhage was experienced; (2) incomplete curettage followed by radium implantation; (3) histological interpretation difficult, but the lesion must now be regarded as an atypical giant-cell tumour (this opinion was expressed by Professor J. C. Bloodgood, who examined the patient and microscopic slides four years ago); (4) no sign of local recurrence after seven years. Presumed complete cure.

Case 11.—H. H. B., male, aged 30. Giant-cell Tumour of Femur, Lower End (R).

History.—Pain and swelling right knee, eight months' duration; symptoms followed definite contusion.

On examination.—(3.10.28.) Swelling and enlargement of internal condyle of right knee with marked local tenderness; slight effusion in joint with limitation of full extension.

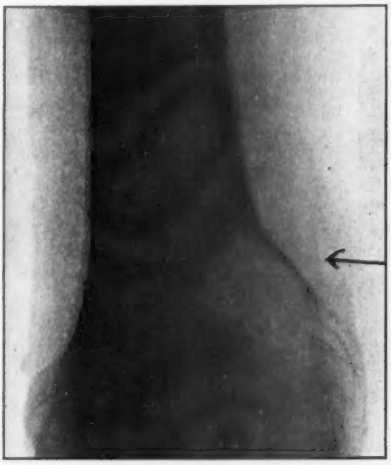


FIG. 18.

FIG. 19.

FIG. 18.—*Case* 11. Cystic expansion of internal condyle with perforation of cortex.

FIG. 19.—*Case* 11. Slight rarefaction in interior of internal condyle with thinning of cortex; a very early stage of a giant-cell tumour.

X-ray.—Shows cystic expansion of internal condyle with slight trabeculation; bone cortex very thin with localized perforation (fig. 18). On further inquiry it appeared that the patient's knee was examined shortly after the injury, and a radiogram obtained which was not available at the time of operation. This radiogram (1.3.28) shows very slight rarefaction in inner condyle with attenuation of cortex (fig. 19).

Treatment.—(5.10.28.) *Exploration: curettage and cauterization.* Cystic area of internal condyle completely filled up by tumour-like tissue, reddish brown in colour; there was also a little clear fluid. Material curetted out and bone cavity carbolized.

Histology.—Typical giant-cell tumour—giant cells abundant, large and multinucleated.

Subsequent course.—(1) Six months later X-ray showed excavated area becoming filled with new bone. (2) One year and one month later, recrudescence of pain and swelling on inner side of knee; slight effusion, with some bulging and marked tenderness over inner condyle.

X-ray.—Considerable reconstruction in inner condyle, but the cortex appeared rather thin and "crinkly." Local recurrence suspected—re-exploration advised.

Treatment.—(3.9.30.) *Re-exploration: curettage and cauterization.* The remains of the bone cavity entered through the old hiatus; the interior was occupied by dense fibrous

tissue containing one or two tiny areas suggesting tumour tissue. The cavity was curetted and cauterized with pure carbolic acid.

Histology.—Spindle-cell tissue and fibrous tissue with no trace of giant cells in any part of the field.

Result.—One year after second operation: no symptoms suggesting recurrence.

Comments.—(1) A rapidly-growing tumour with early perforation of bony shell; (2) effective curettage followed by signs of obliteration of cystic area: histology—typical giant-cell tumour; (3) recurrence of pain and swelling after an interval of nearly two years; (4) re-exploration of the cystic area showed that the cavity was packed with dense fibrous tissue.

These findings suggest that the tumour had healed after the first operation, a view supported by the histology of the material removed at the second operation.

This case illustrates a practical difficulty in the clinical and radiological diagnosis of early recurrence of a giant-cell tumour.

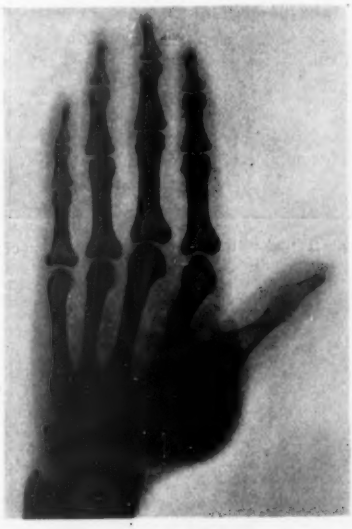


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FIG. 20.—*Case* 12. Giant-cell tumour, first metacarpal. Multicystic expansion and almost complete disappearance of the metacarpal.

Case 12.—M. A., aged 36. Giant-cell Tumour, Metacarpal of Thumb (L).

History—Swelling of left thumb developed without known reason; no record of injury; symptoms eight months' duration.

On examination.—(12.11.28.) Swelling of thumb involving the whole length of the metacarpal bone; skin showed distended veins.

X-ray.—Metacarpal almost completely replaced by tumour with a fairly clear outline; cortex almost completely disappeared (fig. 20). Multicystic appearance with marked trabeculation.

Treatment.—(16.11.28.) *Resection of Metacarpal with Tumour; replacement by bone graft.* Tumour exposed and found to consist of solid tissue, dark red in colour. Owing to complete destruction of the bone shell, curettage was impracticable. The remains of the metacarpal, together with the tumour mass, were excised, leaving the distal articular surface only. After cauterization of the tumour bed a bone graft was impacted into the trapezium and into the cancellous tissue of the remains of the metacarpal head.

Histology.—Typical giant-cell tumour.

Subsequent course.—(9.2.29.) Thumb shows fair function; bone graft appears to be united at each end (fig. 21).

(10.12.29.) No clinical evidence of local recurrence, but X-ray shows rarefaction of bone graft at each end, with ? slight cystic expansion of distal end.

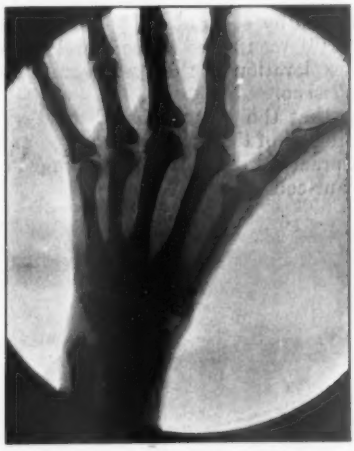

FIG. 21.—*Case* 12. Bone graft in situ three months after excision of metacarpal and tumour.

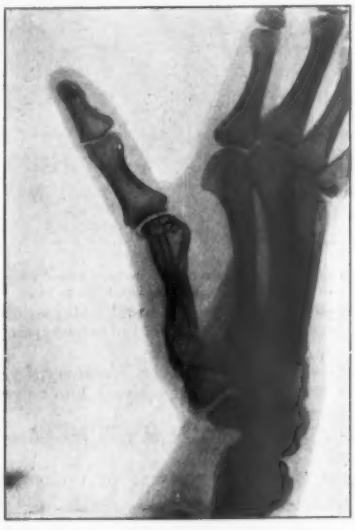

FIG. 22.—*Case* 12. Bone graft honeycombed by a recurrent tumour sixteen months after operation.

March, 1930: Small nodule discovered in region of operation scar; obvious local recurrence.

X-ray.—Shows bone graft honeycobmed; typical multicystic expansion involving the proximal and distal thirds (fig. 22). Further operation advised.

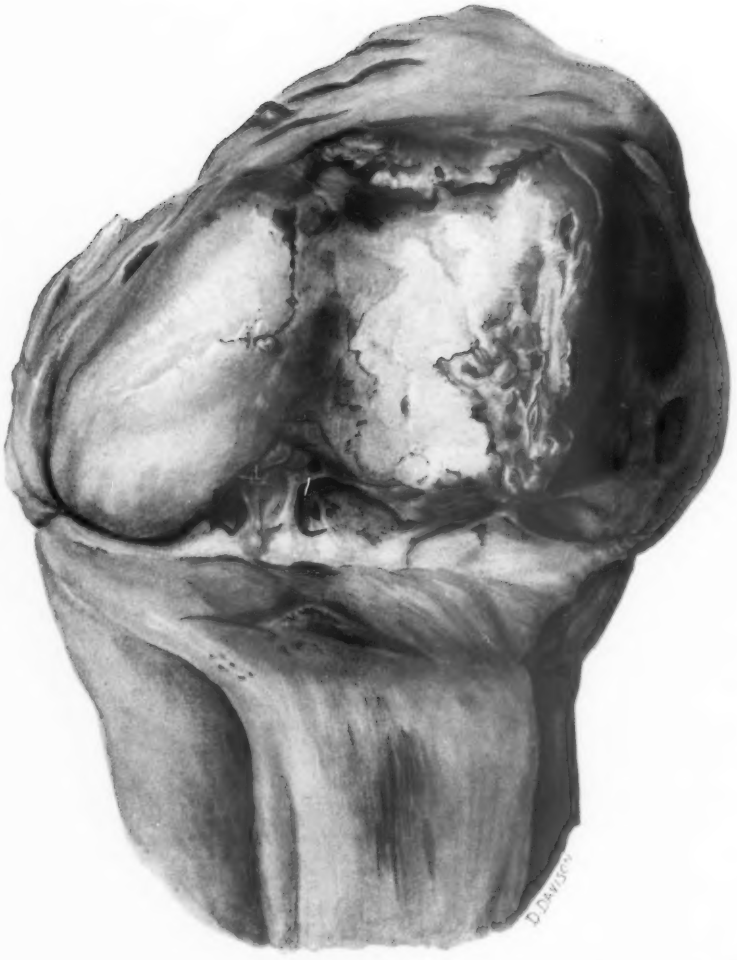

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FIG. 26.—*Case* 18. **Giant-cell tumour of lower end of femur—twelve years' duration—showing impending invasion of the knee-joint.**

Treatment.—(24.3.30.) *Operation*: *removal of bone graft and recurrent tumour.*—The bone graft, which was extensively invaded by tumour growth, was removed entire, together with the trapezium ; outlying nodules of growth discovered invading the adductor muscles ; a clean sweep was made of all tumour tissue and the bed cauterized.

Histology of recurrent tumour. Giant-cell tumour tissue eroding bone (fig. 23).

Result.—One year after second operation : a shortened and somewhat flail thumb, but no evidence of local recurrence.

Comments.—(1) A giant-cell tumour in an unusual situation (metacarpal) ; markedly invasive and destructive ; (2) treated first by excision and cauterization of the tumour bed ; (3) local recurrence with invasion of bone graft used to replace the original metacarpal ; (4) no sign of local recurrence one year after further radical excision.

Case 13.—B. F., male, aged 37. Giant-cell Tumour of Femur, Lower End (L).

History.—Pain and swelling in the left knee for the past twelve years ; history of

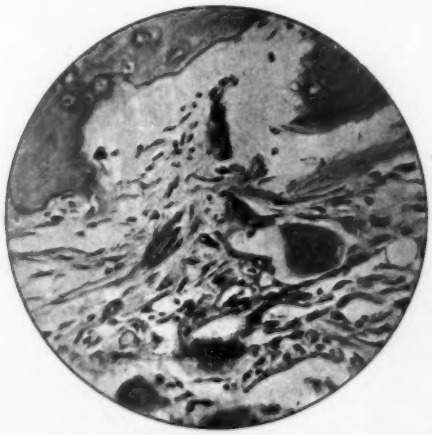


FIG. 23.—*Case* 12. Giant-cell tumour of metacarpal of thumb (L). Giant-cell tumour tissue invading and eroding bone graft. High-power.

repeated injuries ; small operation twelve years ago—nature unknown. Two weeks before reporting at hospital fell on the knee, causing severe pain and disability.

On examination.—(10.3.31.) Marked thickening lower end of femur ; slight effusion in joint ; some limitation of movement ; extreme wasting of musculature of thigh and calf (fig. 24 p. 22).

X-ray.—Shows multicystic expansion lower end of femur ; pathological fracture at upper limit of cystic area ; cortex very thin, especially in the region of articular surface ; coarse trabeculation (fig. 25 p. 22).

Treatment.—(23.3.31.) *Amputation through mid-thigh.*

Description of specimen (fig. 26 see coloured plate).

Histology.—Typical giant-cell tumour with considerable areas of fibrous tissue.

Result.—(Up to date.) Sound amputation stump ; no sign of recurrence.

Comments.—(1) A slowly-growing tumour, attaining considerable size ; bony shell very thin, especially in relation to the knee-joint, where early perforation had occurred ; spontaneous fracture ; (2) amputation necessary for mechanical reasons ; (3) histology of tumour suggests that its invasive qualities are comparatively feeble.

Case 14.—J. H., male, aged 37. Giant-cell Tumour of Tibia, Upper End (R).

History.—Swelling on inner side of right knee followed a series of local injuries.

On examination—(20.1.21.) Fluctuant swelling over inner tuberosity of tibia with definite bony expansion; no interference with function of knee-joint.

X-ray.—Shows cystic expansion inner tuberosity of tibia with perforation of cortex.

Treatment.—(1) (23.1.21.) *Exploration, curettage and cauterization.* An attempt was made to curette the tumour tissue completely; this was found to be impossible owing to tremendous hæmorrhage and the extreme attenuation of the bony shell which separated the knee-joint from the tumour cavity; wound packed; considerable sloughing followed.

FIG. 24.

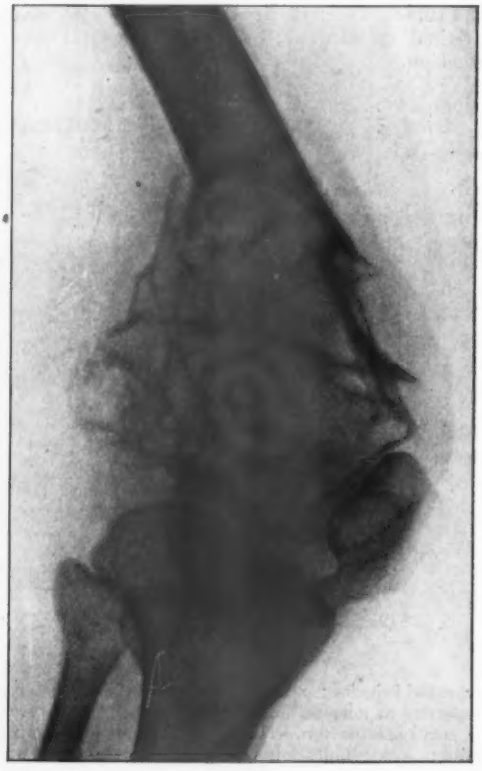


FIG. 25.

FIG. 24.—*Case* 13. Man aged 37. Giant-cell tumour of lower end of femur (L).

FIG. 25.—*Case* 13. Multicystic expansion of lower end of femur with extreme attenuation of the shell, particularly in the region of the articular surface. Pathological fracture at upper limit of cyst.

Three weeks later, owing to the local condition, amputation was advised (fig. 27).

(2) (19.2.21.) *Amputation*, mid-third thigh.

Histology.—Typical giant-cell tumour.

Result.—Ten years later, patient alive and well.

Comments.—(1) Rapidly-growing tumour, markedly invasive, producing early perforation of cortex; (2) complete eradication by curettage impossible without jeopardizing knee-joint; (3) cure by amputation.

FIG. 27.—*Case* 14. Amputation specimen of giant-cell tumour of upper end of tibia; extremely thin shell of bone remaining between the tumour cavity and the knee-joint. *Histology.*—Typical giant-cell tumour.

Case 15.—M. T., female, aged 42. Giant-cell Tumour of Fibula, Lower End (R).

History.—Swelling developed on outer side of right ankle; explored some months later; wound failed to heal and a fungating tumour developed over lower end of fibula.

X-ray.—Shows extensive destruction lower end of fibula.

Treatment.—(1) (24.4.25.) *Exploration.* An attempt was made to remove the tumour mass together with lower third of the fibula; owing to the widespread invasion of the soft parts excision was impracticable. (2) (1.5.25) *Amputation*, lower third of thigh.

Histology.—Typical giant-cell tumour.

Result.—Patient alive and well nine months later. Efforts to trace her during the past six years have been unsuccessful.

Comments.—(1) Giant-cell tumour in an unusual situation (fibula) markedly invasive and destructive, with involvement of soft parts and the production of a fungating tumour; (2) treatment by amputation; (3) ultimate fate unknown, but no reason to anticipate recurrence.

The Sarcoma.

Not the least of the many advantages resulting from the formation of the American Sarcoma Registry, has been the advocacy of a more rational classification of bone sarcomata. The older terminology, in which tumours were variously described according to the predominant cell tissue, consistency, or presumed site of origin, was most unsatisfactory. For the practising surgeon a simple scheme is essential, and preferably one in which the tumours may be presented as clinical types. From this standpoint three main clinical groups of bone sarcoma may be recognized:—

(1) *The osteogenic sarcoma*—embracing the common sarcoma of bone.

(2) *Ewing's sarcoma*—a comparatively rare tumour, which is both a clinical and histological entity, and

(3) *The extra-periosteal sarcoma*—an even rarer tumour which, though more or less accidentally connected with bone, cannot be excluded.

Incidence.—It is said that one case of bone sarcoma is found in every 100,000 inhabitants of the United States of America. The figures for our own population are given as one in 75,000. Hospital statistics again offer some guidance on the problem of incidence. Thus, for example, at St. Thomas's Hospital, during a twenty-one year period, 57 sarcomata of the limb bones were dealt with—an average of 2·7 cases per year (Maybury). It should be noted, however, that in thirteen cases in this series no histological report was available, and many of the patients were beyond the typical sarcoma age-period.

(1) *Osteogenic sarcoma.*—The use of the term osteogenic sarcoma implies the recognition of the common sarcoma of bone as a true osteoblastoma, i.e., a tumour derived from the ancestors of the specific bone cells. This tumour may be further subdivided into various histological types, but as Kolodny has shown, any standardized histological classification is bound to be artificial.

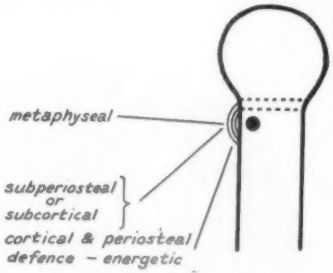
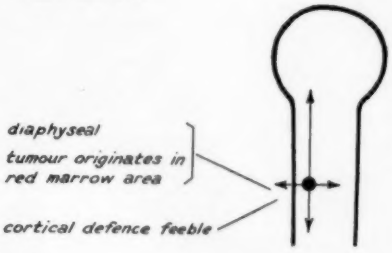
The time-honoured distinction between the *periosteal* and *endosteal* sarcoma also carries little weight, for the average tumour begins as a tiny nodule either under the periosteum, or in the cancellous tissue immediately beneath the cortex. In fact, the true *central* sarcoma producing expansion of bone after the fashion of the giant-cell tumour or benign cyst is extremely rare (Bloodgood)—a fact of considerable diagnostic importance.

It seems likely that future surveys of the large mass of material in the two famous American collections will lead to the differentiation of certain histological types of tumour which have a definite clinical counterpart. This trend is already apparent in some of the most recent contributions of our American colleagues on the subject of bone sarcoma (Phemister, loc. cit.; Geschickter, loc. cit.). For the moment it is wise to reject all elaborate classifications which lack a clinical basis.

Diagnosis.—The broad picture of the osteogenic sarcoma has already been outlined with the tumour posed for comparison with the giant-cell tumour (see Table I). As this picture becomes more deeply ingrained in our sense of clinical perspective, early and accurate diagnosis becomes progressively simpler. With increasing experience it is rarely necessary to invoke the somewhat doubtful assistance of a biopsy, a procedure open to serious criticism in sarcoma of the long bones, but which I believe to be legitimate and useful in tumours in other sites, e.g., the pelvis.

There is little need to dwell on the superficial mimicry of osteogenic sarcoma by such non-tumour lesions as myositis ossificans or syphilitic ostitis. The outstanding problem in the diagnosis of osteogenic sarcoma is provided by the *solitary metastatic tumour of the long bones* in which the primary cancerous growth is latent. In my own experience these secondary growths present themselves at least as often as the

TABLE II.

Osteogenic Sarcoma.	Solitary Metastatic Carcinoma.
Age :—10 – 40 under 35	*Age* :—40 – 60 + over 35
Sex :—M. > F.	*Sex* :—M. = F.
	
X-ray :— Spindle swelling. Erosion of cortex. Radiating ossification. Extra-osseous tumour shadow.	*X-ray* :— Cystic expansion—trabeculated. Destruction of cortex. New bone formation.
	Other bones :—Punched-out areas. Mottling.
Femur ... Lower end. Tibia ... ... Upper end. Humerus ... Upper end. Pelvis.	Femur ... ... Upper ⅓ *shaft.* Humerus ... Upper ⅓ *shaft.* Pelvis. Spine.
Trauma → Pain → Tumour (Fracture 8%)	Pain → Fracture → Tumour ↓ (33%)

sarcomata, and usually in individuals in the prime of life, in good health, and in whom, for some little time, a careful search fails to disclose any sign of primary cancer.

It must be realized that the clinical term, "solitary metastatic growth," may be a misnomer, and that such a tumour may be simply an outward and visible sign of a group of skeletal metastases demonstrable only in radiograms. For this reason, in all solitary malignant growths of bone in patients over the age of thirty-five, a radiographic examination should be made of the major long bones, pelvis, spine and skull. This simple precaution will obviate many mistakes in diagnosis, and patients with secondary tumours will be spared the ordeal of heroic surgical measures appropriate only in a primary growth.

But a metastatic tumour in the humerus or femur may exist as a solitary growth for some time before the advent of general skeletal and visceral dissemination. At this stage the tumour is liable to be regarded as a primary sarcoma, although, as the accompanying table (Table II) will show, the metastatic tumour has its own

distinctive picture. Where the diagnosis on clinical evidence alone remains uncertain, a biopsy is not only justifiable, but likely to give definitive information, owing to the specific histology of metastases derived from primary tumours of the thyroid, breast, kidney (hypernephroma), prostate, or other deep-seated organs.

Case 16.—H. B., female, aged 58. *Secondary Thyroid Deposit in Humerus* (*R*).

History.—Many years of chronic indigestion, due to an hour-glass stomach, regarded as non-malignant on clinical and radiological evidence. Onset of severe pain in the right arm (June, 1929).

On examination.—(18.6.29.) *Right arm.* Marked atrophy of shoulder muscles, with slight tenderness and thickening of the humerus in the upper third.

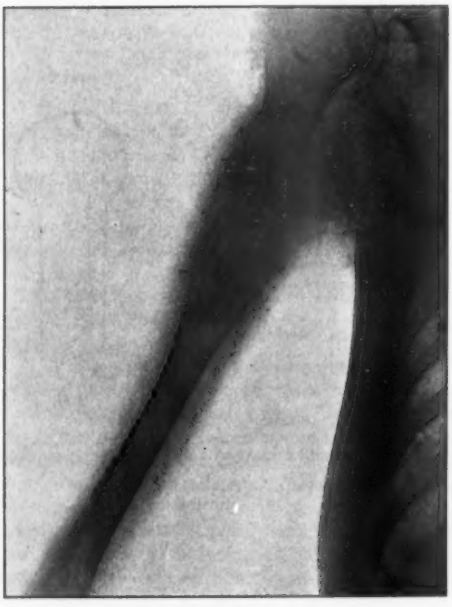


FIG. 28.

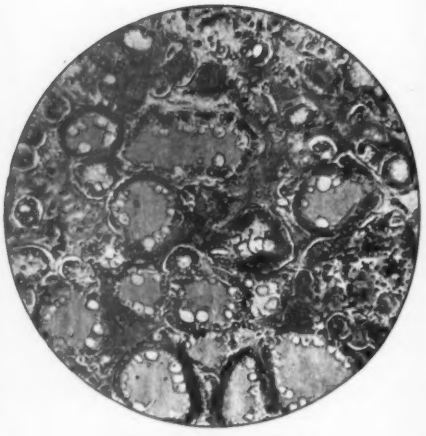


FIG. 29.—High power.

FIG. 28.—*Case* 16. Secondary thyroid-tumour of humerus—showing multicystic expansion in the upper third of shaft with tumour shadow surrounding the bone.

FIG. 29.—*Case* 16. Secondary thyroid deposit on humerus (R). Typical thyroid vesicles containing colloid.

X-ray showed cystic area, with slight expansion in upper third of shaft of humerus (R); well-marked trabeculation. Wassermann test, negative.

Clinical examination revealed no evidence of primary growth; patient averse to treatment of any kind; arm carried in sling.

Subsequent course.—(18.9.29.) Very little change in physical signs. X-ray showed further expansion of bone in cystic area, with attenuation of cortex.

(30.4.30.) X-ray showed increased expansion of multicystic area with definite tumour shadow surrounding the bone (fig. 28). Radiograms of other bones showed no changes suggesting metastases. Operation advised and accepted.

Treatment.—(2.5.30.) *Exploration of humerus: resection of part of shaft with tumour.* A completely encapsuled tumour was exposed, containing greyish red tissue with considerable areas of hæmorrhage. A rapid section made during the operation gave a misleading report, suggesting that the possibility of a primary tumour could not be entirely ruled out.

In view of this a considerable length of the shaft of the humerus was resected with the tumour intact; gap temporarily bridged by a large intramedullary peg.

Histology.—Typical thyroid tissue showing various phases in the evolution of thyroid vesicles; no definite histological evidence of malignancy (fig. 29).

June, 1930.—A re-examination of the thyroid gland revealed the presence of a tiny nodule in the left lobe. Further radiograms of other bones were obtained, and a metastasis was discovered in the ischium on the left side.

Result.—During the eighteen months which have elapsed since the resection of the tumour, the patient's general condition has remained unchanged. The thyroid nodule shows little if any appreciable increase in size. There is no sign of local recurrence of the tumour in the arm, or of visceral metastasis. The secondary deposit in the ischium has increased in size, but no other skeletal metastases have been discovered up to date. Exploration of the thyroid gland has been suggested, but the patient refuses all further treatment.

Comments.—(1) A primary thyroid tumour, latent for some time; (2) a metastatic tumour in the humerus; very slow growth—evidently a low degree of malignancy; tumour temporarily mistaken for primary sarcoma.

Case 17.—P. M. A., male, aged 48. Secondary Deposit in Femur (R). ? Hypernephroma.

History.—Intercostal pain for six months, diagnosed as spinal arthritis; treated by diathermy, etc. Slipped and fell, sustaining a *fracture of the neck of the right femur.* Radiogram at that date showed no bony abnormality in the region of the fracture; treated by fixation and traction. Fracture apparently united at the end of eight weeks and splint discarded. Whilst still confined to bed, felt a sudden snap in the upper part of the right thigh; this was followed by great pain and swelling.

On examination.—(21.8.30.) Marked swelling upper third of right thigh with obvious fracture of the shaft of the femur at this level; traction applied and limb fixed on Thomas' splint.

X-ray showed (1) marked absorption neck of femur with some mottling of surrounding bone; (2) cystic area in shaft of femur at junction of upper and middle thirds.

Wassermann reaction, negative.

A diagnosis of secondary malignant deposit was made, but a clinical examination failed to reveal any evidence of primary growth in the abdomen, thyroid, prostate, or elsewhere.

Subsequent course.—Pain and swelling in thigh gradually increased, and the fracture showed no signs of union. The patient's general condition deteriorated very slowly, and during the next few months there were no clinical signs pointing to the existence of a primary growth. Owing to the continuance of the severe pain and the increasing helplessness of the patient, at his own request the question of amputation of the limb was considered.

X-ray (5.2.31) showed: (1) Cystic expansion of the great trochanter and base of the femoral neck; (2) increase in size of the cyst in upper part of shaft (fig. 30).

Treatment.—(18.2.21.) *Disarticulation at hip-joint (R); insertion of radium.* After a preliminary ligature of the external and internal iliac arteries, the limb was disarticulated at the hip-joint. During the operation it was impossible to avoid cutting through tumour tissue, which was incompletely encapsuled and invaded the surrounding muscles. Fourteen radium tubes were inserted; a blood transfusion was given, and the patient rallied well from the operation.

Histology.—Suggests hypernephroma.

Result.—The amputation flaps healed with a tiny area of sloughing, and the patient was grateful for the removal of his useless and painful limb. Four weeks later he developed anæsthesia and paralysis in the remaining limb; paralysis of the sphincters followed, and he died with symptoms of ascending paralysis. No autopsy available.

Comments.—(1) A metastatic tumour producing spontaneous fracture as the first symptom; (2) primary tumour (? hypernephroma) remaining latent up to the time of death; (3) disarticulation of the limb carried out for the relief of intolerable suffering.

Prognosis and treatment.—From this aspect the story of osteogenic sarcoma makes melancholy reading. It is generally admitted that the outlook is most gloomy in the very young, and more favourable after the age of thirty. We have

little information regarding the prognostic significance of the various histological types of tumour, but the tumours which merit the title of *chondrosarcoma* are said to exhibit a low degree of malignancy (Phemister, loc. cit.). It should be noted, however, that this special group includes the "sequential" sarcomata engrafted on a chondroma or exostosis.

In the treatment of osteogenic sarcoma of the long bones, amputation is still the method of choice, but disarticulation of the limb is necessary only in tumours of the upper end of the humerus or femur. Following amputation, an effort should

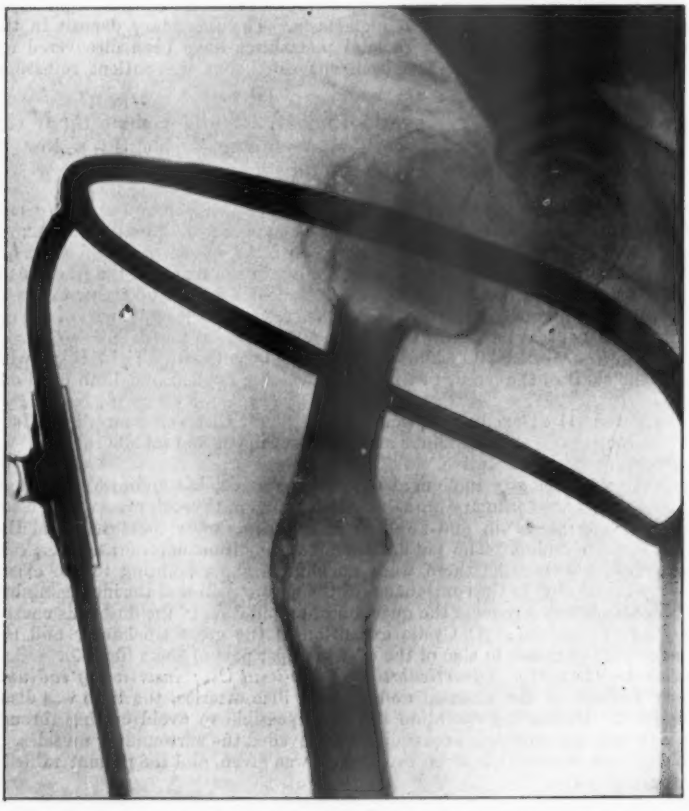


FIG. 30.—*Case* 17. Secondary malignant deposits on femur—? hypernephroma. (i) Cystic expansion in the great trochanter and femoral neck; (ii) a second cyst in the upper third of the shaft.

be made to postpone the inevitable metastases by the administration of Coley's fluid, and deep X-ray therapy applied to the chest at regular intervals. In the majority of osteogenic sarcomata, pulmonary metastases appear within three years from the time of the removal of the tumour. A number of survivals beyond the five-year limit are on record, but in many the histological interpretation is open to criticism. For inoperable tumours there is little to offer beyond a combination of Coley's fluid and radium, the latter a therapeutic agent which in sarcoma is still *sub judice*.

Some of the more important problems relating to the diagnosis, prognosis, and treatment of osteogenic sarcoma, may be further presented in a brief review of my personal experience of this tumour, which is limited to twenty-one cases.

ANALYSIS OF CASES. OSTEOGENIC SARCOMA.

Long bones	16	...	Femur, upper end	2	10
			Femur, lower end	8	
			Tibia, upper end ...	3	
			Fibula, upper end	1	2
			Fibula, lower end	1	
			Humerus, upper end ...	1	
Short bones	1	...	(Cuboid)		
Pelvis ...	3				
Spine ...	1				

				Age.	
Males ...	15	Long bones	...	5 to 30 ...	11
Females ...	6			over 30 ...	5
		Short bones	...	over 40 ...	1
		Flat bones	...	over 40 ...	4

Histological proof 17
No histological proof ... 4

3 over 60 years { 2 diagnosis of sarcoma on clinical and radiological evidence; 1 doubtful (? secondary tumour)

1 aged 22

Treatment:
Amputations 14.—Death under 1 year 7
Death between 1 and 2 years ... 2
Death between 3 and 4 years ... 1
Survival beyond 4 years ... ... 1 (still alive)
Recent amputations (under 1 year) 3 (still alive)
Radium 1.—Shrinkage of tumour—no histological proof.

Group I.—Rapidly growing tumours with early fatal termination.

Case 18.—J. R., male, aged 19. Osteogenic Sarcoma of Upper End of Tibia (R). (Chondro-sarcoma.)

History.—Pain in the right knee of three weeks' duration.

On examination.—(25.1.21.) Swelling and thickening of upper end of right tibia, chiefly on lateral aspect; local tenderness; no alteration in colour of skin; no distension of veins.

X-ray.—(5.2.21.) Showed cavity in upper end of tibia (fig. 31).

Treatment.—(1) (11.2.21.) *Exploration; biopsy.* Cavity entered and found to contain fleshy tissue, pale and mottled. Material removed for histological examination.

Histology.—Osteogenic sarcoma; polymorphous cellular picture with much cartilage formation.

(2) (15.3.21.) *Amputation mid-thigh.* Examination of specimen showed that a large mass of growth had broken through the bone shell posteriorly and extended into the popliteal space; the greater part of the tumour was firm in consistency and showed a considerable amount of cartilage (fig. 32).

Result.—Died with symptoms of pulmonary metatases eight months from the time of amputation. No autopsy available.

Case 19.—J. G., male, aged 15. Osteogenic Sarcoma of Lower End of Femur (L).

History.—Aching pain and swelling, left knee; three months' duration; kick at football six months before.

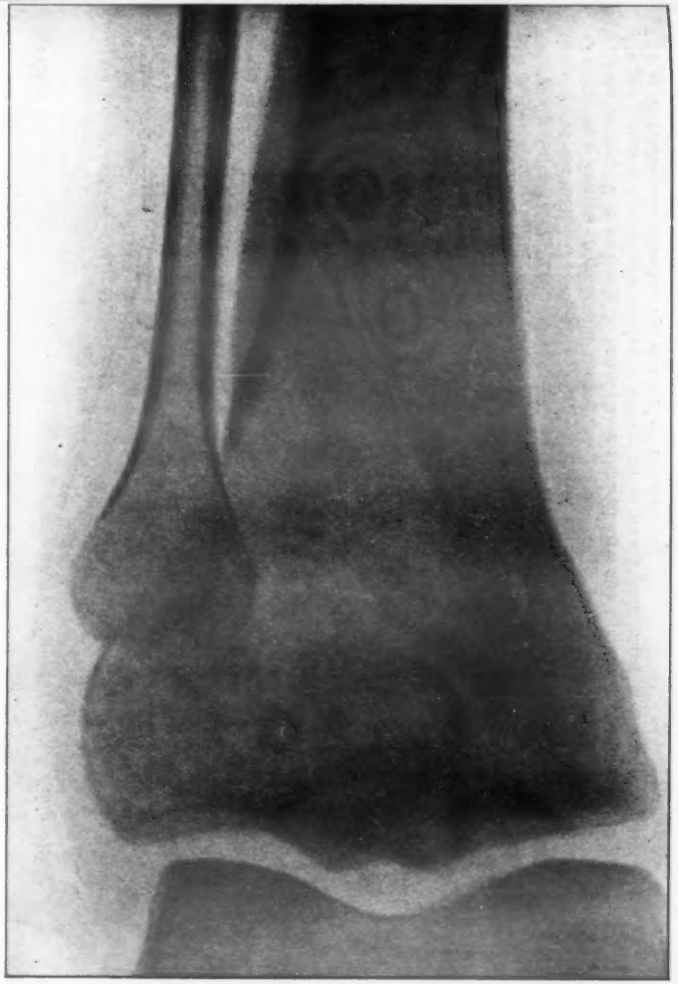
On examination.—(4.12.28.) Marked swelling lower third of thigh, particularly on inner side, with definite thickening of underlying femur.

X-ray showed cavitation on mesial aspect lower end of femur with disappearance and perforation of bony cortex (fig. 33).

Treatment.—(1) (7.12.28.) *Exploration; biopsy.* Bony cavity contained tumour-like tissue of varied consistency and colour. Material removed for histological examination. Rapid section report—? giant-cell tumour.

Section available forty-eight hours later showed that the tumour was a true sarcoma. Amputation was accordingly advised, but for some weeks permission was refused.

(2) (9.1.29.) *Amputation upper third of thigh.*



Histology.—Polygonal cells, large and small, with mitotic figures; no giant cells: ossification from tumour cells (figs. 34 and 35).

Result.—Death with symptoms of visceral metastases *twelve months* after amputation; no autopsy available.

Case 20.—A. B., female, age 25. Osteogenic Sarcoma of Lower End of Femur (L).

History.—Pain in the left knee of three months' duration, with increasing swelling on inner side. Joint forcibly manipulated by unqualified practitioner.

On examination.—(8.1.29.) Marked swelling in region of internal condyle, left femur; obvious bony thickening; marked local tenderness; skin shows dilated veins.

FIG. 33.—*Case* 19. Cavitation on mesial aspect of lower end of femur with disappearance and perforation of cortex.

X-ray showed destruction of lower end of femur on mesial aspect; cavitation, and shadow of tumour extending outside the bone; subperiosteal new bone on posterior aspect.

Treatment.—(11.1.29). *Amputation upper third of thigh.*

Histology.—Osteogenic sarcoma; polygonal cells; giant cells of malignant type.

Result.—Signs of pulmonary metastases four months later; death *five months* from the date of amputation; no autopsy available.

Case 21.—J. M., male, aged 43. Osteogenic Sarcoma of Cuboid (L).

History.—Pain and swelling left foot following injury, eight weeks' duration.

On examination.—(20.1.31.) Large swelling on dorsum of the left foot over the cuboid region; ulceration of skin with blood-stained discharge.

X-ray showed almost complete destruction of cuboid and part of the adjoining cuneiform and bases of outer metatarsals. No clinical evidence of pulmonary metastases, but *radiograms of the chest showed signs of early deposits in the left lung.* In spite of these findings amputation was advised in order to get rid of the tumour which was on the point of fungating through the skin.

Treatment.—(29.1.31.) *Amputation middle third of leg (L).*

Histology.—Osteogenic sarcoma; polygonal cells: ossification from tumour cells.

Result.—Death, with clinical evidence of metastases in lungs and brain *five months* later. No autopsy available.

Here are four rapidly growing tumours with a short clinical history and early visceral dissemination, occurring at different age-periods—in the second, third and fourth decades. Case 18, which conforms to the chondrosarcoma type, showed none of the reputed low-grade malignancy of this tumour. The remaining three showed no histological features of special significance.

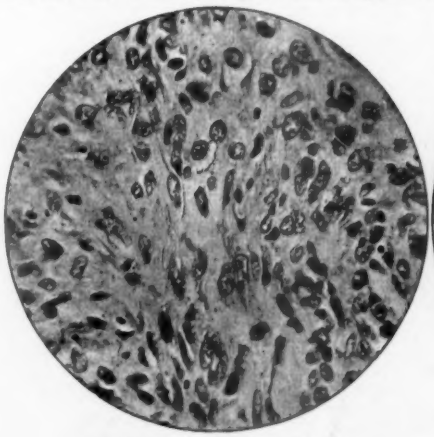


FIG. 34.—High power.

FIG. 35.—Low power.

Case 19. Osteogenic sarcoma of lower end of femur; high power—large tumour cells with mitoses; low power—ossification from tumour cells.

Case 20 is a striking example of the fulminating type of sarcoma. The tumour also involved an unusual bone, the cuboid, and produced extensive local destruction and pulmonary metastases within *eight weeks* from the onset of the first symptom.

Group II.—Tumours showing a long survival period after amputation.

Case 22.—T. L., male, aged 25. Osteogenic Sarcoma of Lower End of Femur (R).

History.—Pain and swelling right knee, four months' duration.

On examination.—(8.2.21.) Right knee swollen with synovial thickening and limitation of movement; some thickening round the lower end of the femur; clinical appearance suggested tuberculous arthritis.

X-ray showed cavitation in interior of lower end of femur, with subperiosteal deposit of bone extending some little distance up the shaft; shadow of tumour material around the bone (fig. 36).

Treatment.—(1) (23.3.21.) *Exploration; biopsy.* Tumour-like tissue exposed in the region of the external condyle. Histology: Osteogenic sarcoma; polygonal cells with malignant giant cells (figs. 37 and 38).

(2) (8.4.21.) *Amputation, upper third of thigh.*

Result.—Patient remained in good health for three years. During this time was treated regularly by injections of Coley's fluid. Re-admitted to hospital three years from the

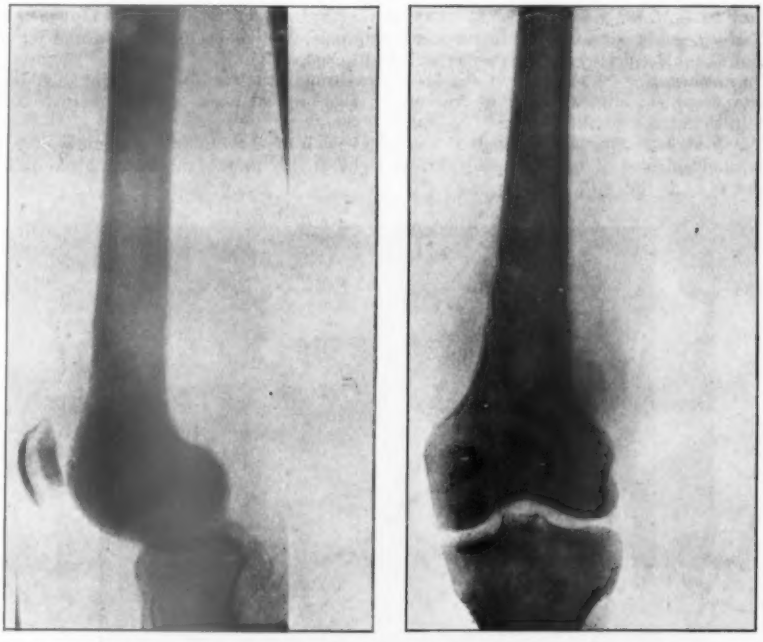


FIG. 36.—*Case* 22. Cavitation in interior of lower end of femur; subperiosteal deposit of bone and shadow of tumour material.

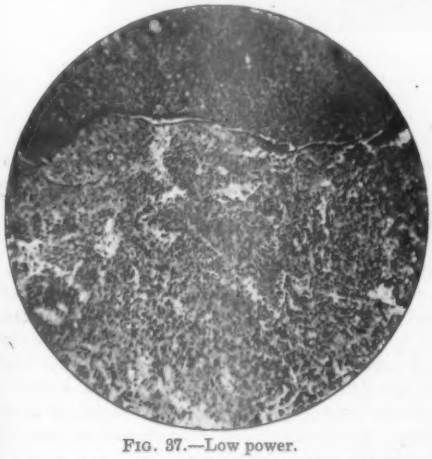


FIG. 37.—Low power.

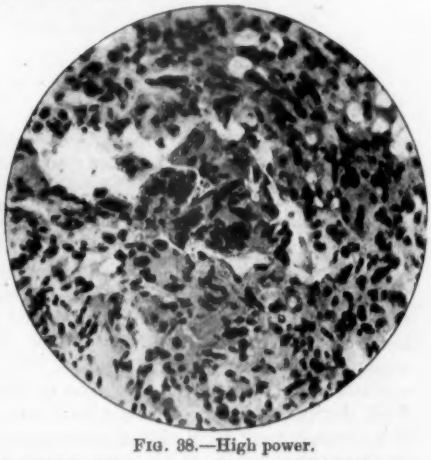


FIG. 38.—High power.

Case 22. Osteogenic sarcoma of lower end of femur (R). Polymorphous cells with malignant giant cells.

date of the amputation with swelling of abdomen and lower limbs, and symptoms suggesting secondary deposits in the chest. Died two months after admission. Autopsy: solitary secondary deposit in wall of left ventricle fungating into the ventricular cavity; no other secondary growths were discovered. Survival after amputation, *three years, two months.*

Case 23.—M. W., female, aged 43. Osteogenic Sarcoma of Upper End of Humerus (R).

History.—Reaching forward to catch a falling pole, felt something snap in the region of the right shoulder; very little pain; reported at hospital the following day.

On examination.—(21.7.27.) Considerable swelling in the region of the right shoulder with definite crepitus; clinical signs of fracture of the surgical neck of the humerus. Dilated veins in the skin over the swelling (fig. 39).

X-ray showed *fracture* through the surgical neck of the humerus; shadow of tumour tissue, and spicules of bone growing out at right angles to the shaft; some cavitation in interior of bone (fig. 40).

FIG. 39.—*Case* 23.—Female, aged 43. Osteogenic sarcoma of upper end of humerus (R). Note the swelling of the right shoulder.

Treatment.—(29.7.27.) *Interscapular-thoracic amputation (R).*

Histology.—Osteogenic sarcoma; polygonal cells, large and small, with malignant giant cells.

Result.—Alive and well four years and three months after amputation; no sign of local or visceral recurrence.

Case 22 is a sarcoma with a conventional histology in a man of 25, showing a survival period beyond the average (three years and two months). At the time of the amputation the tumour had attained a fair size, and had produced a well-marked local destruction of the lower end of the femur. The post-mortem findings were unusually interesting, the visceral dissemination consisting of a single cardiac metastasis and nothing more. The patient was treated by regular courses of Coley's fluid during the three years after amputation, and it is tempting to conclude that this therapy may have played some part in postponing the ultimate dissemination of the tumour.

Case 23 is characterized by a very short clinical history, and the somewhat

unusual feature of a spontaneous fracture as the first symptom in osteogenic sarcoma. The survival period is also considerably beyond the average, viz., four years and three months. But during this time the patient has received no treatment designed to inhibit general metastasis. A reconsideration of the histology of the tumour gives little ground for criticism of the original diagnosis.

The following case, originally included in my series of osteogenic sarcomata, is interpolated as an instructive example of difficulty in histological and clinical interpretation.

Case 24.—T. W., male, aged 27. ? Osteogenic Sarcoma of Upper End of Left Tibia.

History.—Pain in the left knee following injury; onset of swelling with effusion; pain specially severe at night time; symptoms five months' duration.

FIG. 40.—*Case* 23. Fracture through the surgical neck of the humerus; cavitation in interior of bone; shadow of tumour tissue and spicules of bone growing out at right angles to the shaft.

On examination.—(19.2.24.) Swelling over inner tuberosity of tibia with definite expansion of bone; some distension of veins, and œdema of soft tissues.

X-ray showed cavity in inner tuberosity of tibia with clear-cut outline; destruction of cortex on superficial aspect (fig. 41).

Treatment.—(1) (26.2.24.) *Exploration; biopsy.* Periosteum intact but no bony shell remaining; cavity occupied by large fleshy mass consisting of pinkish grey, somewhat friable tissue. Material removed for histological examination.

Histology.—Diagnosis on rapid section: Sarcoma of spindle-cell type (figs. 42 and 43).

(2) (29.2.24). *Amputation upper of third thigh.*

Result.—Patient alive and in good health, with no sign of local or visceral recurrence *seven years and eight months* since the date of amputation. For three years following the operation he had regular deep X-ray applications to the chest, and up to one year ago weekly injections of Coley's fluid.

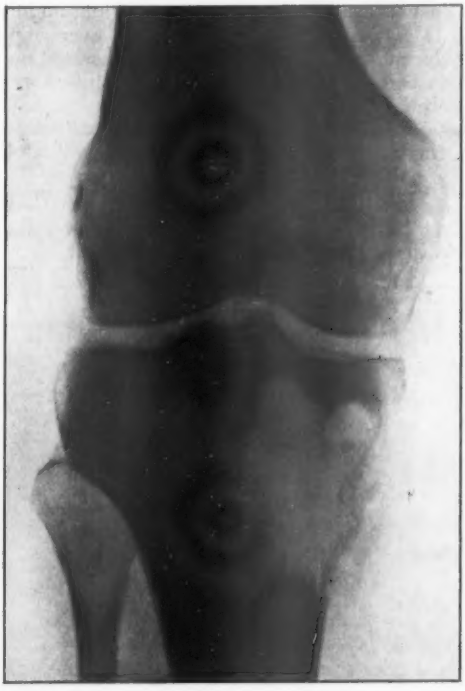


FIG. 41.—*Case* 24. A cavity in the inner tuberosity of tibia with destruction of cortex.

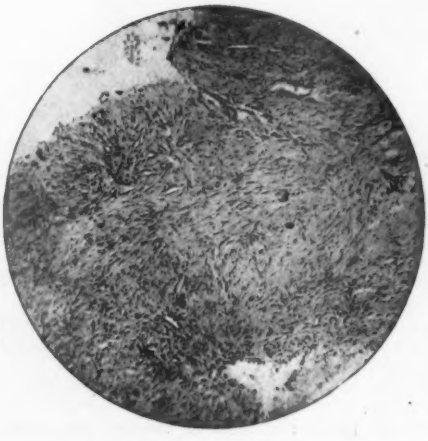


FIG. 42.—Low power.

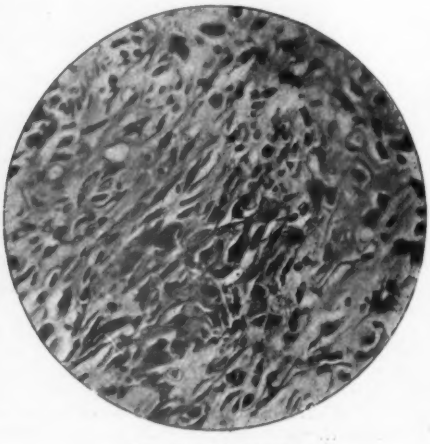


FIG. 43.—High power.

Case 24. ? Osteogenic sarcoma; ? Osteitis fibrosa. Mass of spindle cells with a few small giant cells.

This patient was submitted to Professor J. C. Bloodgood in 1927 as an example of osteogenic sarcoma with an unusually long survival period after amputation, and as illustrating the possible therapeutic value of a combination of Coley's fluid and deep X-ray therapy. Professor Bloodgood regarded the lesion as an atypical localized ostitis fibrosa. He suggested that the cyst in the upper end of the tibia had existed for many years, and that following an injury the lesion had become invasive. The amputation specimen is no longer available, and further microscopic sections made from the original block show nothing more than the spindle-celled histology revealed at the biopsy. This, it must be admitted, is not incompatible with a diagnosis of ostitis fibrosa.

(2) *Ewing's sarcoma.*—To me the Ewing's sarcoma is a will-o'-the-wisp, for I have yet to meet my first example of this clinical and histological entity. At the time of the appearance of Kolodny's monograph, the American Sarcoma Registry contained forty tumours of this type; and there are sixty in the Johns Hopkins collection (Copeland and Geschickter [13]). The somewhat unusual skeletal distribution; the involvement of the *shafts* of the long bones rather than the ends; the tendency to multiplicity; and the clinical picture simulating a chronic inflammatory lesion of bone, are features which distinguish this tumour from the osteogenic sarcoma. Ewing's tumour is believed to correspond to the small round-cell sarcoma of the older terminology, but its exact histogenesis—whether of endothelial or lymphoid origin—is unsettled. It has even been suggested that certain tumours of this type may be metastatic growths (Putti [14]).

The two following tumours, included in my own series of osteogenic sarcomata and involving one of the less familiar bones, the fibula, show a predominant round-cell histology, but do not fulfil the clinical canons of the Ewing's tumour.

Case 25.—A. T., male, aged 5. Osteogenic Sarcoma of Lower End of Left Fibula.

History.—Swelling on outer side of left ankle developed slowly without any history of trauma; symptoms five months' duration.

On examination.—(29.7.27). Swelling overlying the lower third of the fibula consisting of thickening of the soft tissues and of the underlying bone; slightly tender.

X-ray showed outward bowing of lower third of the fibula with localized erosion on mesial surface (fig. 44).

Treatment.—(30.9.27). *Exploration; biopsy; amputation thigh.*—Greyish, friable tumour-like tissue exposed surrounding the lower end of the fibula and burrowing into the adjacent muscles. Material set aside for rapid section; whilst awaiting the report, attempt made to resect affected bone and tumour tissue; this was found to be impossible without jeopardizing the condition of the leg. On the report that material showed histological picture of sarcoma, the limb was amputated through the lower third of the thigh.

Histology.—Sarcoma, chiefly small round-cell type; very little intercellular substance (figs. 45 and 46).

Result.—Symptoms of pulmonary metastases six months later. Death *one year* from the date of amputation; no autopsy available.

Comments.—A rapidly-growing tumour in a young child, in an unusual site (fibula), with early dissemination and fatal ending.

Case 26.—J. D., male, age 21. Osteogenic Sarcoma of Fibula, Upper End (R).

History.—Twisted right knee whilst playing football; swelling developed on outer aspect; six weeks later reported at hospital.

On examination.—(11.7.27). Swelling over upper end of right fibula with induration of soft parts, and definite bony thickening.

X-ray showed subperiosteal new bone round the neck of the fibula with slight erosion of the cortex.

Treatment.—(14.7.27). *Exploration; biopsy; amputation mid-third of thigh.*

An encapsuled tumour was exposed surrounding the neck of the fibula and containing large masses of granulation-like tissue. Material sent for rapid histological examination. On the report, sarcoma round-cell type, the limb was amputated through the middle third of the thigh.

Histology.—Sarcoma. Cells closely packed; many of round-cell type.

Result.—Patient developed clinical signs of lung metastases six weeks later, and died three months from the date of amputation.

Autopsy.—Metastatic deposits in lungs showing histology of primary tumour.

Comments.—A small tumour with a fulminating course, approximating to the round-cell type.

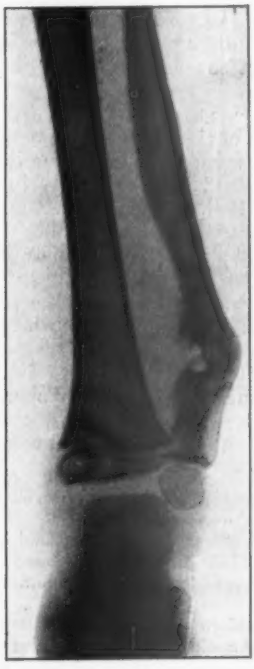


FIG. 44.—*Case* 25. Outward bowing of the lower third of the fibula with erosion on mesial aspect.

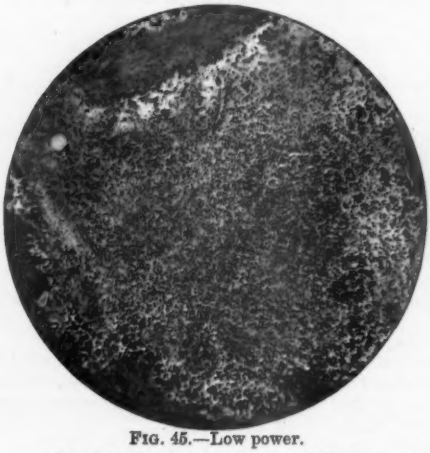


FIG. 45.—Low power.

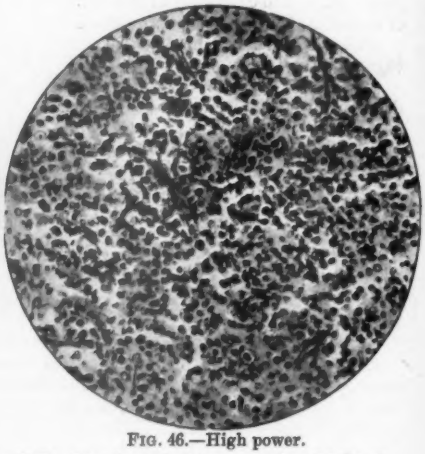


FIG. 46.—High power.

Case 25. Osteogenic sarcoma of fibula, lower end (L). Closely packed tumour cells, chiefly of the round-cell type.

(3) *Extraperiosteal sarcoma.*—This term is applied to tumours originating in the outer layer of the periosteum, which belong to the *fibro-sarcoma* group. The tumours are well encapsuled, grow slowly, and do not invade the underlying bone until a very late stage. The prognosis after excision or amputation is much more favourable than in osteogenic sarcoma, and long periods of survival may be prophesied. These characteristics are illustrated in the two extraperiosteal sarcomata from my own series.

FIG. 47.

FIG. 48.

FIG. 47.—*Case* 27. Extraperiosteal sarcoma of femur (L). Shows large swelling of middle third of left thigh.

FIG. 48.—*Case* 27. Tumour shadow around the shaft of the femur; slight cortical thickening, but no evidence of bony destruction.

Case 27.—E. S., female, aged 24. Extraperiosteal Sarcoma of Femur (L).

History.—Swelling in thigh discovered three weeks ago; gradually increasing in size. No history of injury.

On examination.—(10.8.26.) Large swelling in middle third of left thigh; firm on palpation, and apparently attached to the shaft of the femur over its middle two-thirds; skin shows distended veins (fig. 47).

X-ray showed tumour shadow around the shaft of the femur with a little cortical thickening, but no evidence of bony destruction (fig. 48). Wassermann test, negative.

Treatment.—(1) (20.8.26.) *Exploration of tumour; biopsy.* Tumour was well encapsuled, and consisted of dense greyish tissue. Histology.—Preliminary histological diagnosis: spindle-celled sarcoma (figs. 50 and 51).

(2) (30.8.26.) *Disarticulation of hip-joint.*

Description of specimen (fig. 49).

Result.—Alive and in good health with no sign of local or visceral recurrence *five years and two months since amputation.*

Comments.—A typical fibro-sarcoma of enormous size, arising in the periosteum of the shaft of the femur; survival beyond the five-year limit.

FIG. 49.—*Case* 27. Amputation specimen; enormous tumour, encapsuled, and incorporated with the periosteum of the shaft of the femur.

FIG. 50.—Low-power.

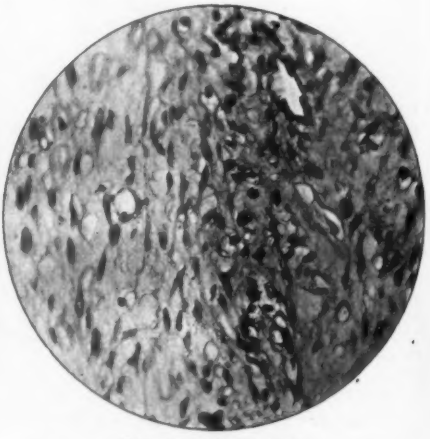


FIG. 51.—High-power.

Case 27. Extraperiosteal sarcoma of femur (L). Spindle cells closely packed.

Case 28.—W. S., male, aged 43. Extraperiosteal Sarcoma of Metacarpal of Thumb (R).

History.—Swelling right thumb some months' duration, increasing slightly in size.

On examination.—(5.7.27.) Small tumour mass about the size of a walnut occupying the dorsum of the thumb, overlying the distal part of the metacarpal and interphalangeal joint; tumour fixed to bone; skin shows distended veins.

X-ray showed slight cavitation in end of metacarpal with shadow of tumour surrounding the head of the metacarpal and proximal phalanx.

Treatment.—(22.7.27). *Disarticulation of thumb—carpo-metacarpal joint.* An attempt was made to excise the tumour, which was found to be closely incorporated with the periosteum of the metacarpal and phalanx, and tendon sheaths. Amputation was accordingly performed at the carpo-metacarpal joint.

Histology.—Sarcoma—polymorphous cells—spindle cells predominant.

Result.—No sign of local or visceral recurrence *four years and three months* since amputation.

Comments.—A typical extraperiosteal sarcoma involving an unusual bone; moderately long survival period.

I am indebted to my colleague, Dr. Arnold Renshaw, for the majority of the histological preparations used in this paper; also to the staff of the Radiological Department of the Ancoats Hospital for unstinted coöperation.

The drawings of tumour specimens are by Miss D. Davison, of Manchester.

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Discussion.—Mr. NAUGHTON DUNN said that he had had a case of Ewing's tumour three years ago. A boy had been admitted into Wolverhampton Hospital because of pain in the thigh. Skiagrams showed a thickening of the bone, with new layers of periosteal bone, and the case presented a picture like that of osteomyelitis. The pain in the thigh was accompanied by a rise in temperature to 101° or 102° F., lasted for about a week, about every third week. The femur had been opened and the cortex removed at that hospital on a diagnosis of osteomyelitis. The skiagram suggested, however, that the site of the infection had been missed. He (Mr. Dunn) admitted the boy to Coleshill Hospital, and explored the suspected infected area. There was a thickened cortex, and underneath some greyish material; and there appeared to be a cystic lining, or a fibrous tissue lining; it looked like osteomyelitis. He closed the wound, and the boy went away. He remained well six months. He (Mr. Dunn) had regarded the condition as osteomyelitis of the femur which had responded to operation and healed up. Six months later the boy returned with pain in the lower part of the femur. Every three weeks there was a recurring temperature, up to 101° or 102° F., and skiagrams suggested a chronic periostitis with osteomyelitis. He excavated the lower end of the femur, which was found to be full of the greyish material already mentioned. A specimen of this was sent for examination, and the report was that it was an endothelial tumour of bone.

The boy remained well for fifteen months, and was then re-admitted with head symptoms, and died a few weeks later.

Mr. C. MAX PAGE said that in one interesting group shown, that of osteoclastoma, a point in the treatment was routine curettage and cauterization. Was the cauterization effected by the use of carbolic? If so, what strength was used? Had there been any recurrences afterwards?

Had the President used radium in the treatment of other giant-celled tumours? If so, what results followed? In the literature he (Mr. Max Page) had not found evidences of the degree of radio-sensitiveness of these tumours. He had himself treated three by radium, and, so far apparently effectively, but they had only been under observation for two or three years.

The President seemed to be relatively optimistic about the treatment of osteogenetic sarcoma with Coley's fluid and deep X-ray therapy applied to the chest at intervals. Coley's fluid had been on the market twenty-five years, and his own view was that it had no effect on osteogenetic sarcoma; possibly he had not yet seen it properly applied. He had submitted one patient to lead treatment after an amputation of the leg for sarcoma of the tibia. After each use of it the patient was very ill, and death took place two years after the operation.

Mr. H. A. T. FAIRBANK said that early in the present year he had had in hospital a woman, aged about 20, a typist, complaining of pain in the humerus, of a few months' duration. A skiagram showed rarefaction of the upper half of the shaft of the humerus, extending into the head. A difference of opinion had arisen as to whether the condition was malignant or inflammatory. There was a spontaneous fracture which was proceeding to unite. He explored it without finding pus, but some soft growth, pale in colour, was present inside the bone. An attempt was made to cultivate this, but it was sterile. After some hesitation the pathologist said that in his opinion it was malignant. The patient twice was treated with radium, and the condition had now cleared up entirely, within the short space of six months. All that remained was a small clean-cut notch beside the humerus, possibly caused at operation.

His own small experience of traumatic subperiosteal sarcoma had led him to believe that it was very malignant. He remembered the case of a boy who was struck on the tibia by the door of a motor car, and was dead within four months. Another patient, a girl aged 17, struck her shin against an iron stove, and two or three months later had a small swelling on the subcutaneous surface of the tibia. A section from it was reported to be sarcoma. Amputation was carried out through the thigh. Two years and a half afterwards there was no sign of malignancy. One pathologist suggested that in this case the tumour had arisen in nerve tissues. He (the speaker) did not know the reason for that suggestion; could the President give any information on the subject?

Recently he had the case of a woman, aged 70, who fell and sustained a fracture of the shaft of the femur, in its upper third. Two or three months later he saw her because union had not taken place, and he advised carrying on with the splint. Two or three months later still she had a tumour of tremendous size, with two fractures, and much rarefaction. On referring back to the early and intermediate skiagrams, there was nothing to cause suspicion of growth. One would not have regarded it as a spontaneous or pathological fracture. The late Professor Shattock said that he had seen only one case in which he was convinced that sarcoma had developed in callus after fracture.

The PRESIDENT (in reply) said that with reference to the treatment of the giant-celled tumour or osteoblastoma by curettage and cauterization, he meant scooping out the contents of the cyst and then swabbing it with pure carbolic acid. That was the original technique introduced by Bloodgood many years ago, and he (the President) had found it satisfactory in cases in which the destruction had not proceeded so far that conservative treatment was out of the question. Some of the cases which he had shown to-night were neglected tumours which had caused considerable local destruction, and in those complete removal of the tumour by curettage was not to be contemplated.

He had never used radium for giant-celled tumours, except in the case of the doubtful tumour in the posterior part of the ilium to which he had referred.

His experience of Coley's fluid in the treatment of sarcoma had been very small, but some importance must be attached to the results obtained by Coley himself. Coley's view was that these toxins actually had a systemic effect, and were worth using with the idea of killing sarcoma cells which were on the point of producing secondary tumours. He admitted that treatment by Coley's fluid was very distressing, and that very few patients would submit to it for long. Still there was nothing else one could offer a patient after amputation.

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